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A MANUAL
OF
PATHOLOGICAL ANATOMY

BY
CARL ROKITANSKY, M.D.
CURATOR OF THE IMPERIAL PATHOLOGICAL MUSEUM, AND
PROFESSOR AT THE UNIVERSITY OF VIENNA, ETC.

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THE
PATHOLOGICAL ANATOMY
OF
THE ORGANS OF RESPIRATION
AND
CIRCULATION.

TRANSLATED FROM THE GERMAN

BY

GEORGE E. DAY, M.D., F.R.S.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS:
PROFESSOR OF MEDICINE IN THE UNIVERSITY OF ST. ANDREWS, ETC.

CONTENTS OF VOL. IV.

PART I.

ABNORMAL CONDITIONS OF THE RESPIRATORY ORGANS.

| | PAGE |
|---|------|
| I.—ABNORMAL CONDITIONS OF THE AIR-PASSAGES | 3 |
| § 1. Deficiency and Excess of Formation | 3 |
| § 2. Deviations in size | 4 |
| <i>a.</i> Morbid Dilatations of the Air-passages | 4 |
| 1. Dilatation of the Larynx and of the Trachea | 4 |
| 2. Dilatation of the Bronchi (Bronchiectasis) | 5 |
| <i>b.</i> Contraction of the Air-passages | 12 |
| <i>c.</i> Hypertrophy and Atrophy | 13 |
| § 3. Deviations in Form | 14 |
| § 4. Deviations in Position | 14 |
| § 5. Interruptions of Continuity | 15 |
| § 6. Diseases of Texture | 15 |
| <i>A.</i> Diseases of the Mucous Membrane and of the subjacent Areolar Tissue | 16 |
| <i>a.</i> Hyperæmia and Anæmia | 16 |
| <i>b.</i> Inflammations of the Mucous Membrane | 17 |
| 1. Catarrhal Inflammation | 17 |
| <i>a.</i> Acute Catarrhal Inflammation | 17 |
| <i>β.</i> Chronic Catarrhal Inflammation | 17 |
| 2. Exudative Processes (Croupous Inflammation) | 17 |
| 3. Pustular Inflammation | 22 |
| 4. The Typhous Process on the Mucous Membrane of the Air-passages | 22 |
| <i>c.</i> Inflammation of the Sub-mucous Areolar Tissue | 25 |
| <i>d.</i> Ulcerous Processes | 26 |
| <i>e.</i> Edema of the Mucous Membrane of the Air-passages | 27 |
| <i>f.</i> Gangrene of the Air-passages | 28 |
| <i>g.</i> Adventitious Products | 28 |
| <i>B.</i> Diseases of the Cartilaginous Skeleton of the Air-passages | 28 |
| <i>a.</i> Inflammation of the Perichondrium of the Laryngeal Cartilages (Perichondritis Laryngea) | 28 |
| <i>b.</i> Inflammation and Softening of the Epiglottis | 29 |
| <i>c.</i> Ossification | 29 |
| <i>d.</i> Adventitious Products | 30 |
| § 7. Anomalies of the Contents of the Air-passages | 35 |

| | PAGE |
|---|------|
| II.—ABNORMAL CONDITIONS OF THE PLEURA | 38 |
| § 1. Deficiency and Excess of Formation | 38 |
| § 2. Anomalies in the Size and Form of the Pleural Sacs | 38 |
| § 3. Diseases of Texture | 39 |
| <i>a.</i> Hyperæmia of the Pleura | 39 |
| <i>b.</i> Inflammation of the Pleura (Pleuritis) | 39 |
| <i>c.</i> Adventitious products | 46 |
| § 4. Morbid Contents of the Pleural Sacs | 48 |
| III.—ABNORMAL CONDITIONS OF THE LUNGS | 50 |
| § 1. Deficiency and Excess of Formation | 50 |
| § 2. Anomalies of Size—Hypertrophy and Atrophy | 50 |
| § 3. Anomalies in Form and Position | 53 |
| § 4. Diseases of Texture | 54 |
| <i>a.</i> Rarefaction of the Pulmonary Tissue—Emphysema | 54 |
| <i>b.</i> Condensation of the Pulmonary Tissue | 61 |
| <i>c.</i> Hyperæmia, Stasis—Apoplexy of the Lungs | 62 |
| <i>d.</i> Anæmia of the Lungs | 69 |
| <i>e.</i> Edema of the Lungs | 69 |
| <i>f.</i> Inflammations of the Lungs (Pneumoniz) | 71 |
| 1. Croupous Pneumonia | 72 |
| Typhous Pneumonia | 88 |
| 2. Catarrhal Pneumonia | 89 |
| 3. Inflammation of the Interstitial Tissue of the Lungs—Inter- stitial Pneumonia | 90 |
| <i>g.</i> Deposits in the Lungs—Metastatic Processes | 91 |
| <i>h.</i> Gangrene of the Lungs | 94 |
| <i>i.</i> Softening | 98 |
| <i>k.</i> Adventitious Products | 99 |
| SUPPLEMENT. | |
| § 1. Diseases of the Thyroid Gland | 124 |
| § 2. Diseases of the Thymus Gland | 127 |

PART II.

ABNORMAL CONDITIONS OF THE ORGANS OF CIRCULATION.

| | |
|---|-----|
| I.—ABNORMAL CONDITIONS OF THE PERICARDIUM | 131 |
| § 1. Deficiency and Excess of Formation | 131 |
| § 2. Deviations in Size and Form | 132 |
| § 3. Interruptions of Continuity | 132 |
| § 4. Diseases of Texture | 133 |
| <i>a.</i> Inflammation | 133 |
| <i>b.</i> Secondary Formations | 138 |
| § 5. Anomalies of the Contents of the Pericardium | 140 |

CONTENTS.

ix

| | PAGE |
|--|------|
| II.—ABNORMAL CONDITIONS OF THE HEART | 141 |
| § 1. Deficiency and Excess of Formation | 142 |
| § 2. Anomalies of Form | 147 |
| § 3. Anomalies of Position | 148 |
| § 4. Anomalies of Size | 150 |
| a. Abnormal Size | 153 |
| b. Abnormal Smallness | 169 |
| § 5. Anomalies of Consistence | 171 |
| § 6. Separations of Continuity | 171 |
| § 7. Diseases of Texture | 175 |
| a. Hyperæmia, Anæmia | 175 |
| b. Inflammations | 175 |
| 1. Inflammation of the Lining Membrane of the Heart—Endocarditis | 175 |
| Hypertrophy and Atrophy of the Endocardium | 189 |
| 2. Inflammation of the Muscular Substance of the Heart—Carditis (in the strict sense of the word, Myocarditis) | 191 |
| Aneurism of the Heart | 195 |
| c. Metastasis in the Muscular Substance of the Heart | 203 |
| d. Gangrene of the Heart | 203 |
| e. Adventitious Products | 203 |
| ABNORMAL CONDITIONS OF THE VALVES, AND ESPECIALLY OF THEIR OSTIA. | |
| § 1. Deficient and Excessive Formation | 225 |
| § 2. Anomalies of Size.—Hypertrophy and Atrophy of the Valves | 225 |
| § 3. Anomalies of Form | 230 |
| § 4. Anomalies of Consistence | 230 |
| § 5. Separations of Continuity | 232 |
| § 6. Diseases of Texture | 232 |
| a. Inflammation (Endocarditis) of the Valves | 232 |
| Aneurism of the Valves | 236 |
| b. Adventitious Structures | 239 |
| Review of the Anomalies of the Valves, and more especially of those producing Contraction of the Ostia and Insufficiency | 241 |
| SUPPLEMENT. | |
| Cyanosis | 243 |
| III.—ABNORMAL CONDITIONS OF THE ARTERIES | 251 |
| § 1. Deficiency and Excess of Formation | 251 |
| § 2. Anomalies in the Origin, Course, &c. of the Arteries | 251 |
| § 3. Anomalies and Diseases of Texture | 252 |
| a. Inflammation of the Arteries—Arteritis | 252 |
| b. Ulcerous Processes | 260 |
| c. Excessive Deposition of the Lining Membrane of the Vessels | 261 |
| d. Adventitious Products | 273 |
| § 4. Anomalies of Calibre | 275 |
| a. Dilatation of the Arteries (Aneurism) | 275 |
| Aneurism of the Aorta | 296 |
| Dilatation of the Ductus Botalli | 298 |

| | PAGE |
|---|------|
| Traumatic Aneurism | 299 |
| Hernial Aneurism | 300 |
| B. Abnormal Narrowness, Contraction, and Obliteration of the Arteries | 303 |
| § 5. Mechanical Separations of Continuity | 313 |
| A. On Laceration of the Larger Arteries | 313 |
| Dissecting Aneurism | 313 |
| B. On Incised, Penetrating, and Gun-shot Wounds of the Arteries | 322 |
| Spurious Aneurism | 322 |
| Varicose Aneurism | 323 |
| The Process of Healing and Obliteration after an Artery has been cut through or tied | 327 |
| IV.—ABNORMAL CONDITIONS OF THE VEINS | 335 |
| § 1. Excess and Deficiency of Formation | 335 |
| § 2. Anomalies in their Origin and Course | 335 |
| § 3. Diseases of Texture | 335 |
| a. Inflammation | 335 |
| b. Hypertrophy of the Venous Coats, especially of the Lining Mem- brane | 355 |
| c. Adventitious Structures | 359 |
| § 4. Anomalies of Calibre | 361 |
| A. Dilatation (Phlebectasis) | 361 |
| B. Occlusion, Contraction, and Obliteration of Veins | 374 |
| § 5. Separations of Continuity | 376 |
| SUPPLEMENT. | |
| ANOMALIES OF THE SMALL VESSELS AND CAPILLARIES. | |
| § 1. Anomalies of Calibre, and especially Dilatation | 377 |
| § 2. Separation of Continuity | 379 |
| § 3. Anomalies of Texture | 379 |
| Capillary Phlebitis | 380 |
| Excessive Depositions of the Lining Membrane | 382 |
| Adventitious Products | 382 |
| V.—ABNORMAL CONDITIONS OF THE LYMPHATIC SYSTEM | 382 |
| A. The Lymphatic Vessels | 383 |
| § 1. Anomalies of Texture | 383 |
| a. Inflammation | 383 |
| b. Adventitious Products | 386 |
| § 2. Anomalies of Calibre | 387 |
| B. The Lymphatic Glands | 387 |
| § 1. Anomalies of Volume.—Hypertrophy, Atrophy | 387 |
| § 2. Anomalies of Texture | 389 |
| a. Inflammation | 389 |
| b. Acute Swellings of the Lymphatic Glands | 392 |
| c. Adventitious Products | 393 |
| § 3. Anomalies of the Contents of Lymphatic Glands | 398 |

PART I.
DISEASES OF THE RESPIRATORY SYSTEM.

4

PART I.

ABNORMAL CONDITIONS OF THE RESPIRATORY ORGANS.

WE shall consider the abnormal conditions of the respiratory organs under three heads :—

- 1st. As they occur in the air-passages ; namely, the larynx, trachea, and bronchi.
- 2d. As they occur in the pleuræ ; and—
- 3d. As they occur in the lungs.

The deviations from the normal state occurring in the thyroid and thymus glands will be noticed in a Supplement.

I.—ABNORMAL CONDITIONS OF THE AIR-PASSAGES.

§ 1. *Deficiency and Excess of Formation.*

An entire deficiency of the air-passages invariably occurs in cases in which the lungs are absent. A partial deficiency, as for instance of the trachea, may occur without the lungs being necessarily absent, the bronchi in such cases being given off directly from the larynx. Under this head we must notice imperfect development of the air-passages, dependent on the deficiency of certain parts entering into their structure, as of some of the laryngeal cartilages or tracheal rings ; and their malformations arising from the arrest of development both in length and width.

An excess of formation is seen in the duplication occurring in double monsters, in which either the upper or the lower portion of the pulmonary apparatus is doubled ; and here we must also place the occurrence of a supernumerary third bronchus, occasionally noticed on the right side in persons otherwise normally developed. Finally, we sometimes meet

with supernumerary laryngeal cartilages and tracheal rings, thereby increasing the distance to the tracheal bifurcation.

2. *Deviations in Size*.—In noticing the *calibre* of the air-passages, we shall omit the consideration of those cases in which the whole apparatus is either extremely developed; or where, on the other hand, it appears in an undeveloped condition with its walls in an attenuated state, and shall at once proceed to the subject of *acquired dilatations and contractions*. In noticing the *thickness of the walls* of the air-passages, we shall have to consider the *hypertrophy and atrophy* of the various structures entering into their composition.

a. *Morbid Dilatations of the Air-passages*.—These occur in variously recurring forms in the larynx, trachea, and bronchi, and sometimes occasion a dilatation of the *whole* apparatus; much more commonly, however, the dilatation affects only *single portions* of the respiratory organs, as, for instance, the bronchi, where, by the way, this morbid change is most frequently noticed.

1. *Dilatation of the Larynx and of the Trachea*.—An uniform dilatation of this canal is not unfrequently seen in *marasmus* or *senile atrophy*. Its existence in advanced age is interesting, since it always occurs with senile marasmus of the lungs (*emphysema senile*), and is more or less proportional to it. They are both dependent on the wasting of the tissues entering into the formation of the larynx and trachea.

There is another form of dilatation, which proceeds from *hypertrophy and relaxation of the posterior wall of the trachea, with or without saccular or hernial protrusion of the mucous membrane*. This form is extremely rare in the larynx, as, indeed, might have been naturally expected from the protected state of the interstices between its different cartilages; in fact, we scarcely ever see even a tendency towards it: while in the trachea it is of frequent occurrence, and is sometimes developed to an astonishing degree. Although bronchial dilatations have been well understood since the time of Laennec, little has been observed in reference to dilatation of the trachea, and especially in regard to this form.¹

¹ See Oesterr. Jahrb., vol. xvi, p. 3.

In the first place, there is a relaxation of the posterior wall of the trachea, giving rise to a great augmentation of surface, especially in the lateral directions. Moreover, its mucous membrane, transverse muscular fibres, and mucous glands increase in bulk, and the excretory ducts of these glands become dilated; while, on the other hand, the elastic, yellow, longitudinal fibres become attenuated and disappear. If protrusion of the mucous membrane should now occur, it gradually makes its way between the thickened transverse fibres in the form of a cleft or funnel, and finally of a transversely-placed, saccular expansion, usually deepest at the posterior part of the tracheal rings, where we find a distorted, cleft-like orifice of the excretory duct of a mucous gland. The larger this hernia or false diverticulum becomes, so much the more prominently do the muscular bands, which limit it, project on the inner surface of the trachea; and here, if the herniæ be numerous and close upon one another, the muscular fibres form a lattice-work, in which the cross bars are usually single, but occasionally bifurcated at one of the extremities.

This condition is dependent on repeated and chronic catarrhs of the trachea, and forms *one* of a number of analogous cases occurring in other parts of the body. In saccular dilatation, the hypertrophied mucous glands on the posterior wall of the trachea may, by the traction through the medium of their ducts, draw the tracheal mucous membrane between the bundles of transverse fibres. These dilatations sometimes extend along the whole trachea, and even into the bronchi.

These dilatations of the trachea closely correspond with the similar dilatations of the bronchi, proceeding from hypertrophy and paralysis.

2. *Dilatation of the Bronchi (Bronchestasis).*—There are forms of bronchial dilatation besides that which depends on the wasting of the tissues in old age, or senile marasmus. In fact, this portion of the air-passages is remarkable for the frequency with which dilatations occur, and for the degree of development which they attain. They constitute one of the most important diseases of the air-passages.

There are *two forms* of bronchial dilatation which especially claim our attention:

a. In the *first* we find a bronchial tube *uniformly dilated*

through a certain extent; that is to say, the dilatation has taken place uniformly at all points of its periphery, so that a tube, which in the normal state will admit only of a fine probe, will now admit of the passage of a crow- or goose-quill, or even of a larger body. The dilatation is very striking and distinct, when we see a bronchial tube far exceeding in size the stem from which it is given off. It is seldom confined to a single tube, but, as a rule, affects a distinct portion of the bronchial tree; and its branches and twigs may either undergo an augmentation proportional to their relative natural sizes, or, as is more frequently the case, the dilatation becomes more considerable the deeper and further we proceed. In this it observes a law to which we shall frequently recur.

β. The *second* form is the *saccular dilatation*. Here we find a bronchial tube dilated into a fusiform or roundish sac; the dilatation in the latter case very frequently preponderating in such a direction, that the greater space of the bronchial sac lies altogether out of the axis of the tube entering or leaving it. These sacs, in rare cases, attain the size of a hen's egg, but most commonly they are of the size of a bean, hazel-nut, or walnut. We find, also, that either one or several bronchial tubes may undergo this saccular dilatation, while on both sides of the sac the normal calibre is retained, or the whole bronchial ramification may be affected. In the latter case, numerous similar sacs of various sizes are so arranged, that, collectively, they form a large ramifying sinuous cavity, whose individual excavations are bounded and separated from one another by ridge-like or valvular duplicatures, projecting inwards from the bronchial walls. Saccular dilatation of the bronchial extremities constitutes a special variety, which is frequently observed in the form of thin membranous vesicles, completely filled with air, and occurring, either singly or in groups, in the vicinity of cicatrising tubercle in the apices of the upper lobes. One or more bronchial tubes, in taking their course through the impermeable substance of the apices of the upper lobes, crowded with obsolete and cretified tubercles, and, as it were, saturated with pigment, become compressed by the shrivelled parenchyma, and are finally obliterated; their extremities then expand into the above-mentioned vesicles; and, according to the state of the bronchial tubes—whether they are merely compressed or

actually obliterated—the sacs may be emptied by gradual pressure, or will resist all attempts to expel the air.

Of these two forms of bronchial dilatation, the *second* appears to be the more common, and in young persons is undoubtedly the more frequent. The *degree* of dilatation may be determined by the proportion which it bears to the size of the bronchial tube, and to the calibre of the parent stem, from which it is given off. From the observations we have already made, it is obvious that, while its *extent* may be very limited, it may on the other hand be so considerable, that all the bronchial tubes of one lobe, or even of a whole lung, may be thus affected.

Bronchial dilatation occurs, for the most part, in the smaller tubes, and, as a general rule, is most frequent and, at the same time, most extensive, in those of the third and fourth order. It is never found in the two primary bronchi, or, at all events, very rarely, and then in the same form as the tracheal dilatations already described.

The bronchi near the surface and borders of the lungs are most liable to this affection; and this fact may be regarded as one of the evidences (and there are others) of the affinity between this affection and true vesicular emphysema of the lungs. The upper lobes are the most common seat of bronchial dilatations.

The walls of the dilated bronchi are found in various conditions. Sometimes we observe the mucous membrane and the fibrous sheath *hypertrophied* and *thickened*. The former appears in a state of chronic catarrh, being tumid, of a more or less dark-red tint, of a loose spongy appearance, and permitting of being easily torn. The bronchi are rigid; on making a section of the lung, they appear like wide, gaping tubes, from which a thick, yellow, purulent mucus is seen to flow, and their white, thick fibrous sheaths strongly contrast with the inner layer of tumid and reddened mucous membrane. Such is the usual character of the first form of bronchial dilatation.

In the saccular form of dilatation, the walls are *relaxed* and *attenuated*. The mucous membrane of the bronchial sacs is only slightly, or not at all, reddened; it is more commonly pale; the firmness of its tissue is very little, or not at all, modified;

and it generally presents a smooth and polished appearance, similar to that of a serous membrane. The sacs contain a thin, pale-yellow, puriform fluid, or an almost colourless vitreous mucus.

From the very striking and almost constant differences presented by the bronchial walls in these two forms of dilatation, we are led to infer that there are corresponding differences in their nature and their causes; and we shall presently have an opportunity of pointing out in what these differences actually consist.

The pulmonary tissue surrounding a bronchial dilatation is generally of increased density, and finally becomes obliterated. We shall subsequently enter fully into the consideration of this change, and the conditions giving rise to it; and shall point out its importance in the establishment of a theory relating to the production of bronchial dilatation.

Laennec, and almost all subsequent pathologists, believe that bronchial dilatation is always a mechanical consequence of catarrh; that it is dependent on the accumulation of the bronchial secretion at certain points, and the powerful inspirations made during paroxysms of coughing, whereby the walls, from some unknown causes, become at one time thicker, and at another thinner, than natural. The condensation of the surrounding parenchyma is, according to their views, simply dependent on the compression exercised by the dilated bronchus, which thus becomes the primary cause of the final obliteration of the pulmonary tissue. Hence, according to Laennec, the bronchial dilatation is the primary anomaly, and the condensation of the pulmonary substance is merely a secondary change dependent on its compression by the tube.

Corrigan has recently published a theory to account for bronchial dilatation, which is directly the opposite to that of Laennec; and believing the disease to be allied, in its anatomical elements, to cirrhosis of the liver, he has given it the name of *cirrhosis of the lungs*. He regards the atrophy and obliteration of the parenchyma of the lung as the *primary* phenomenon in the development of the disease, and as occurring spontaneously, while the dilatation of the bronchi is *consecutive*, and is not only dependent on the tendency to fill the space thus rendered vacant, and on the expansion occurring in the

act of inspiration, but also on the traction exerted on the opposite walls of the bronchial tubes, by the shrinking of the surrounding tissue.

To speak more precisely, the change that the parenchyma of the lung surrounding bronchial dilatations undergoes, consists, in extreme cases, of obsolescence and destruction of the cellular spaces, and of the contraction of the cells so as to form a cellulofibrous or even a callous and fibro-cartilaginous tissue, which may be either white or streaked with blackish-grey pigment, dotted or of an uniform colour, and which is so intermixed with the fibrous sheath of the bronchus that the two form one continuous whole.

When we take into consideration the vast extent to which the parenchyma of the lung around a dilated bronchus is obliterated; when we reflect on the nature and the degree of this metamorphosis, which very rarely follows prolonged external pressure on a lung; and, finally, when we notice the circumstance that this metamorphosis does not always develop itself uniformly around the dilated tube, nor is most marked the nearer we approach it; we cannot help doubting if all this can be produced and explained by the mere pressure caused by the dilatation of a thin membranous bronchial tube.

In point of fact, on instituting a closer examination, we arrive at conditions of another kind, which are in themselves sufficient to explain this metamorphosis, and are of the greatest importance in reference to the genesis of bronchial dilatation.

Whichever be the form under which bronchial dilatation appears, bronchitis must be regarded as the most frequent primary cause. It acts in different ways, but not mechanically, from accumulation of mucus, according to the theory of Laennec.

In the *first* form of bronchial dilatation, as we have described it in a preceding page, atony and paralysis of the contractile and irritable elements of the tubes are present, dependent on chronic inflammation and blennorrhœa. The facility with which the walls undergo dilatation through the influence of the inspirations and the concussion induced by the paroxysms of cough, is proportional to the amount of exertion required to throw off the secretion accumulated in the bronchial tubes. Moreover the circumstance that many of the smaller tubes are completely

obstructed by the blennorrhœal secretion, favours the above condition. This form of bronchial dilatation affects that portion of the bronchial system in which blennorrhœa occurs.

The *second* or saccular form of bronchial dilatation is not developed in that portion of the bronchi which is the seat of catarrh, but beyond it; it is the consequence of bronchitis in the final ramifications of the bronchi, and depends on their obstruction by the accumulation of secretion, on the tumid state of their mucous membrane, and finally on their actual obliteration. It is produced by the hinderance that is presented to the free ingress of the inspired air, and is proportional to the difficulty of breathing (Reynaud), and the prolonged length of each individual inspiration; and it is especially developed in and about the perfectly impermeable bronchial tubes. The parenchyma surrounding this portion of the bronchial system collapses, and thus produces a space which becomes filled by the dilating bronchus. The dilatation thus lies entirely, or, for the most part, in a collapsed and apparently compressed portion of parenchyma; hence, the latter appears to be the *primary* anomaly, and the bronchial dilatation merely a *resulting* and consecutive morbid change. This circumstance, together with the fact that the collapsed parenchyma passes into the above-described state of complete obliteration, and thus as it were contracts upon itself and causes additional space in the lung, closely approximates this theory to that of Corrigan.

According to him the primary affection is not bronchitis, but is a disease of the parenchyma, not so much presenting the characters of inflammation of the interstitial areolar tissue, as of a pneumonic process (of which we shall subsequently treat) insidiously extending itself from one lobule to another, and depositing a product which becomes indurated, and fused or blended, as it were, with the original tissue. The air-cells become thus obliterated and destroyed, and undergo the same change. The resulting cellulo-fibrous tissue may here, as in the first case, draw asunder the bronchial walls by the tension induced by its further contraction, and may thus contribute to the further dilatation of the bronchial sac and to the increased attenuation of its walls.

However this may be, a smaller or larger proportion of the lungs always become obsolete and shrivelled in proportion to

the extent of the bronchial affection ; indeed, when the bronchial tubes of a whole lung are thus affected, we find that all of its parenchyma is more or less obliterated, contracted to a small part of its normal volume, as if in consequence of external pressure from exudation, and drawn up in the mediastinum towards the bronchus; moreover, the cavity of the thorax is diminished, in consequence of the sinking of its walls over the cellulo-fibrous tissue surrounding the dilated bronchial tubes.

In some rare cases a bronchial sac is entirely separated by obliteration, not only from its own branches, but also from the tube on which it was situated. It then exhibits the appearance of a perfectly closed cavity, which, in consequence of the persistent secreting activity of its lining membrane, is probably further enlarged by the accumulation of mucus. Subsequently, however, if this secreting action be suspended, the accumulated matter becomes inspissated and diminished in volume, till ultimately there is nothing left but a fibrous capsule, enclosing either a soft, fatty, calcareous mass, or a solid concretion of bone-earth.

Isolated saccular dilatations containing puriform matter may be mistaken for *tuberculous cavities*, especially when they are associated with pulmonary tubercles and are situated in the upper third of the superior lobes of lung,—the ordinary focus of pulmonary tuberculosis. On a closer examination we may recognise a saccular bronchial dilatation by the roundish form of the cavern and its pouches, by the smooth, uninjured, investing mucous membrane, by the absence of all signs of ulceration in the bronchial tubes entering it, by the striking difference between its contents and tuberculous pus, by the circumstance that the surrounding and contiguous obsolete parenchyma contains no tuberculous granulations, or only such as are obsolete, and by the simultaneous occurrence of similar cavities in parts of the lung, in which tuberculous excavations are not usually found. In other cases our diagnosis must be founded on general principles, as for instance, on the fact that bronchial dilatations are ordinarily found in the superficial parenchyma of the lungs towards their borders, and very rarely occur in the summit; and further, that, when the bronchial disease is very extensive, tuberculosis is incompatible with it.

In consequence of the obliteration of a large extent of lung produced by extensive bronchial dilatation, we find that this affection gives rise to a development of the right side of the heart in the form of active dilatation, stasis and dilatation of the whole venous system, cyanosis, and vicarious development of the permeable portions of the lungs, which not unfrequently leads to bronchial and pulmonary hæmorrhage (*hæmoptoic infarctus*). If the bronchial dilatation be very highly developed, it induces collapse, emaciation, a cachectic appearance, dropsy, and finally total exhaustion.

In consequence of the venosity and cyanosis to which it gives rise, it affords a very striking immunity, not only from pulmonary tubercles, but from tuberculosis in general. The fact that bronchial dilatation exerts an excluding influence on pulmonary tuberculosis has been known since the time of Laennec; and although the reasons for this influence are not understood, it has served, in recent times, as the basis of several plans for the cure of pulmonary consumption.

b. Contraction of the Air-passages.—This may occur in any part of the respiratory apparatus; but the nature and degree of the affection may be extremely various; in fact, in the latter point of view, the change may proceed to closure and perfect obliteration.

1. It may be dependent on *external pressure*. There may be contraction of the larynx and of the trachea from an enlarged thyroid gland; of the trachea and bronchi from enlarged lymphatic glands, aneurisms, large cancerous deposits in the neck and mediastinum, enlarged thymus glands, and effusions into the cavity of the chest; and of the left bronchus by a dilated left auricle (King). In this manner the air-tubes become forced in various directions from their normal position, and their calibre, as may be seen in contraction of the trachea, may be so encroached on as to represent a mere fissure, having a transverse, an antero-posterior, a straight, or a crescentic form.

2. Contraction may be the result of *disease of the mucous membrane, or of the subjacent mucous tissue* of the air-passages, as of hypertrophy, inflammatory swelling, or œdema of the mucous membrane or of the submucous areolar tissue, of various excrescences, cancerous deposits, or cicatrices after loss of substance; the most frequent cause, however, is bronchitis,

especially when it has given rise to obliteration of the finer bronchial tubes.

3. The calibre of the air-passages may be diminished by *foreign bodies* of various kinds which have penetrated into them either from without or from the intestinal canal through the pharynx or morbid openings; and by products of morbid processes in the mucous membrane and in the deeper tissues, as in adjacent organs, which abnormally communicate with the air-passages; amongst such products we may enumerate coagula of blood, clots of mucus, frothy bronchial secretion, croup-membranes, pus, masses of tubercle, fragments of cancerous matter, acephalocysts, pieces of necrosed cartilage and bone, &c.

c. Hypertrophy and Atrophy.—We have already spoken of hypertrophy of the mucous membrane of the air-passages, of the muscular fasciculi of the trachea, of the fibrous sheaths of the bronchial tubes in cases of dilatation arising from catarrh and blennorrhœa, and of hypertrophy of the mucous follicles in the posterior wall of the trachea, when that canal is the seat of dilatation; it now remains for us to consider more especially *hypertrophy of the mucous membrane and of its follicles in the larynx and the trachea.*

When only moderately developed it presents the ordinary characters of hypertrophy of mucous membranes. In a higher degree it especially affects the mucous glands, and in the larynx gives rise to glandular swelling of the mucous membrane at those points where the glands are most abundant, as for instance, on the superior vocal chords, in the ventricles, over the transverse muscle, and on the epiglottis. In the trachea we observe, in the swollen mucous membrane of the posterior tracheal wall, the dilated mouths of the excretory ducts of mucous glands, lying behind the muscular layer. These glands enlarge to the size of a hempseed, or even to that of a pea or cherry, and become converted into simple, or sinuous, imperfectly partitioned sacs, in whose cavities there is an accumulation of a whitish, opaque, or transparent and vitreous mucus.—In its highest degree the glandular swelling of the laryngeal mucous membrane degenerates *into polypoid hypertrophy, or into cellular or mucous polypi.*

Atrophy exhibits itself in the form of a wasting of the mucous membrane and glands of the air-passages, especially in the

larynx and trachea, a deficiency of mucous secretion, and, at the same time, attenuation of the laryngeal muscles, and is followed by the dilatation of the larynx, trachea, and bronchi which we have already noticed as peculiar to old age.—The epiglottis is sometimes the seat of atrophy and relaxation, arising apparently from slow inflammation; or, on the other hand, it may become indurated and variously misshaped, and thus rendered insufficient for its duties. Further, we must here mention the frequent cases of attenuation and final absorption of the laryngeal cartilages, and the tracheal and bronchial rings with their intervening membranes, arising from the pressure of superimposed tumours, and especially of aneurisms.

3. *Deviations in Form.*—Here we must mention the acquired malformations to which the larynx, trachea, and bronchi are subject, occurring in the form of flattening, indenting, or curving from a morbid and enlarged thyroid gland, an encysted tumour, or an aneurism, or in consequence of cicatrisation after destructive ulceration.

The epiglottis is especially liable to present remarkable anomalies, being found irregularly flattened, and with its edges immoderately sloped or bent backwards, turned down, or rolled together like a horn. These malformations are either the consequence of the contraction of cicatrices in its mucous membrane, submucous areolar tissue, or its actual substance, or of inflammation of its cartilage, with consecutive softening or induration and atrophy of its substance, converting it into a rigid fibrous tissue.

4. *Deviations of Position.*—These principally occur in the larynx and trachea, which may be forced from their perpendicular direction in the neck to either side of the vertical line by partial enlargement of the thyroid gland, by encysted tumours on the side of the neck, by aneurisms, abscesses, cancerous deposits, or wry neck; they may be displaced forwards by swollen and inflamed cervical vertebræ, and by abscesses seated in them; whilst they may be thrust backwards into the dorsal curvature of the vertebral column by aneurisms of the arch of the aorta, cancerous deposits in the anterior mediastinum, &c.

The occasional but rare dislocations of the laryngeal articulations, must also be mentioned as a cause of deviation of position.

5. *Interruptions of Continuity.*—We must here take into consideration :—

a. The various injuries of the air-passages from cutting or stabbing instruments, gun-shot wounds, fractures and minor injuries of the hyoid bone and laryngeal and tracheal cartilages; also lacerations in consequence of forcible concussions or contusions; and, finally, injuries arising from the entrance from without of angular and pointed foreign bodies into the aforesaid air-passages.

b. The gradual solutions of continuity in consequence of atrophy, especially induced by the continuous pressure of aneurisms; and—

c. The numerous separations of continuity dependent on various ulcerous processes acting either from within outwards, or in the opposite direction.

All of these, more or less rapidly, give rise to abnormal communications between the air-passages and the surrounding areolar tissue, and (continuing their progress externally) connect the former with the adjacent cavities and canals, as with the pleural sacs, blood-vessels, or œsophagus, or with abscesses in the lungs, bronchial glands, vertebræ, or lateral and anterior walls of the thorax, allowing not only the passage of air in various directions from the respiratory organs, but also the far more perilous entrance of blood, purulent and ichorous fluids, food and drink into them.

(We might here consider the congenital cervical fistulæ described by Dzondi, Ascherson, Serres, and others. They are certainly anomalies of original formation: but we still require more precise information regarding their mode of development and their signification.)

6. *Diseases of Texture.*—Diseases of texture occur in all the tissues entering into the composition of the air-passages, but especially in the mucous membrane, which is the primary seat of disease in by far the greatest number of cases and in the greatest variety of form. Hence its diseases claim the most particular attention. They frequently extend to the subjacent tissues, and, for the most part, lead to their destruction.

1. Diseases of the Mucous Membrane and of the Subjacent Areolar Tissue.

a. Hyperæmia and Anæmia.—*Hyperæmia* of the air-passages is of comparatively common occurrence. When seated in the finer bronchial ramifications, it is combined with hyperæmia of the parenchyma of the lung; but in the larger bronchial tubes, and in the trachea and larynx, it usually exists alone, and independently of that affection. Hyperæmia varies extremely in importance, according as it is active, or simply mechanical and dependent on an obstructed circulation, or passive, which is a more rare affection; constituting, in any case, an independent disease, which finally gives rise to *hæmorrhage*, or, under other conditions, to *stasis*, and thus to acute or chronic *inflammation*. To the first belong the hæmorrhages from the mucous membrane of the bronchi, trachea, larynx, and epiglottis. On examining the dead body, we find the air-passages to a certain degree filled with coagulated or fluid blood, and patches of the mucous membrane swollen, of a dark-red colour, bleeding when pressed, and apparently loosened in texture; we find no other source of hæmorrhage, no pulmonary apoplexy, nor mechanical or ulcerous separation of continuity. The lungs, as we find in other hæmorrhages from the air-passages, present a dark or light-red speckled appearance from the deposition of blood in the terminations of the bronchial tubes and in the air-cells; but at the same time are (elsewhere) emphysematous, swollen, and pale, in consequence of the obstructed condition of the bronchial tubes, and the impediment thus presented to free expiration.

These hæmorrhages occur in the active form, during the period of evolution, and when there is general plethora, as vicarious to menstrual and hæmorrhoidal fluxes. They arise from and accompany the congestions which so frequently precede the development of tubercles in the lungs, and may be produced by any strong exertion, but especially by the over-taxing of the respiratory organs. They may arise from any sudden shock to the lungs, from the sudden rarefaction of the atmosphere, and they are very frequently dependent on mechanical hyperæmia, resulting from hypertrophy and dilatation of the heart.

Anæmia of the mucous membrane of the air-passages is more or less developed in senile and other varieties of atrophy.

b. Inflammations of the Mucous Membrane.

1. *Catarrhal Inflammation.*—This is one of the most common diseases of the air-passages. It may be either *acute* or *chronic*, and in one or other of these forms often attacks only single portions of the whole bronchial apparatus. In the acute form, however, it not unfrequently extends over the whole bronchial tract, and in the chronic form also is sometimes as widely diffused, but in this case the inflammatory action is more intense at some points than at others.

We distinguish according to their positions, *catarrhs of the larynx, trachea, and bronchi*; or *laryngitis, tracheitis, and bronchitis catarrhosa*.

a. Acute Catarrhal Inflammation.—This presents the same anatomical appearances in whatever part of the bronchial system it occurs; there are various degrees of redness, relaxation, and swelling of the tissue, which, according to the intensity and stage of the affection, secretes a diminished or increased amount of muco-serous, frothy fluid (*sputum crudum*), or thick whitish or yellowish puriform mucus (*sputum coctum*), or, finally, of true purulent matter (the transition to superficial suppuration). The swelling of the mucous membrane and the submucous tissue, which assume the form of watery infiltration, from the areolar tissue being accumulated at individual spots, is important and worthy of great attention, on account of the facility with which it interferes with the calibre of the tubes. This swelling of the mucous membrane is most dangerous when it affects the epiglottis, the folds limiting the glottis, the covering of the vocal chords, and the portion lining the ventricles: the danger is less, but still very great, when it attacks the smaller bronchial tubes.

Acute bronchial catarrh, when widely diffused, is an important, and in children a perilous disease, not only in consequence of the contraction and perfect impermeability of the bronchial tubes caused by the swelling of the mucous membrane and the accumulation of its secretion, but from its occasionally, and especially in children, extending to the air-cells, forming catarrhal pneumonia.

β. Chronic Catarrhal Inflammation.—This is very frequent

in certain portions of the air-tubes; it is often remarkable for its great intensity, and is of the highest importance from its sequelæ. These observations especially apply to chronic bronchial catarrh, but the affection is also common in the larynx and trachea, and sometimes extends over the whole course of the air-tubes; it is generally, however, then especially developed in some one particular part. It possesses the usual anatomical characters of chronic inflammation of mucous membranes; but as no acute catarrh is so liable to frequent relapses, and exhibits such a tendency to become habitual as that affecting the air-passages, so also the chronic form is here especially liable either to relapse into acute inflammation, with an augmentation of intensity, or, on the other hand, to degenerate into *blennorrhœa*. It gives rise to swelling of the mucous membrane, especially on those parts of the larynx which we have already described as abounding in glands, thus causing *glandular hypertrophy*, *mucous polypi*, and *cauliflower epithelial growths*; similarly in the trachea, and more especially in the bronchi, it causes a *spongy thickening* of the mucous membrane; and these affections may lead to *hypertrophy and relaxation of the submucous muscular strata, of the fibrous portions of the vocal apparatus, and of the fibrous sheaths of the bronchi*, and sometimes to *ulcerous destruction*, especially of the larynx, in the form of *diffuse catarrhal suppuration*, or of *catarrhal follicular ulceration*.

Chronic catarrh may further give rise to *diminution* of the calibre of the air-passages, amounting even to their perfect closure; the previous loss of substance sometimes inducing adhesion and perfect *obliteration* of the bronchial tubes. At other times, associated with hypertrophy and paralysis of the tissues, it gives rise to bronchial *dilatation*.

The quantity of whitish, cream-like, or yellow purulent secretion that is thrown off by the bronchial mucous membrane in a state of blennorrhœa, is very remarkable, especially where dilatation is at the same time present. The cases of what is termed Phthisis pituitosa (asthma humidum, bronchial blennorrhœa), fall under this head; and on making an incision through the lungs, abundance of mucus is seen gushing out of the divided bronchi, and pouring over the cut surface.

Both the acute and the chronic form of pulmonary catarrh,

may occur as isolated and substantive diseases; they are, however, frequently associated with catarrhs of other mucous membranes. The acute is frequently of an exanthematous nature, and seems especially connected with measles, smallpox, and typhus; while the chronic form is often of a gouty, scrofulous, or syphilitic nature, and is associated with the most different pseudo-plastic processes on the mucous membrane, and in the submucous tissues. It is the chronic form of bronchial catarrh which accompanies pulmonary tuberculosis, especially true tuberculous phthisis. Moreover, it very frequently arises from mechanical hyperæmia induced by cardiac diseases.

Gonorrhœal catarrh of the larynx requires especial notice, in consequence of its sequelæ. In the form of gonorrhœal metastasis, it attacks the mucous membrane of the epiglottis, and the lateral duplicatures of the glottis and of the superior vocal chords, converting the mucous membrane and subjacent areolar tissue into a fibro-lardaceous, white, resistant structure of considerable thickness, thus giving rise to contraction of the rima glottidis and the cavity of the larynx. This constitutes *gonorrhœal stenosis of the larynx*.

There are two distinct modes in which chronic bronchial catarrh acts injuriously on the parenchyma of the lung. It sometimes causes emphysema; at other times collapse and obliteration of the air-vessels, and consequently obliteration of the pulmonary tissue itself. Amongst its sequelæ we may mention livor, cyanosis, active dilatation of the right side of the heart, and hydrothorax; and the patient dies asphyxiated through some of these affections, or sinks from tabes under the form of Phthisis pituitosa.

2. *Exudative Processes (Croupous Inflammation).*—Under this head we must place processes allied to each other, since they originate in one general disease, but differ extremely in their local morbid centres. This difference exhibits itself anatomically in the physical qualities of the inflammatory products on the free surfaces of mucous membranes, and in the condition of the mucous membrane itself, and of the submucous areolar tissue. These processes, especially in true croup, are primary and independent; or in their collective forms they may be secondary affections—the evidence of a degenerated acute or chronic disease.

True croup, the exudative process yielding a plastic, fibrinous product, claims our first attention. We scarcely ever observe the primary, genuine croupous process to occur anywhere except on the mucous membrane of the air-passages, where it appears as *laryngeal, tracheal, or bronchial croup*, ordinarily known as *laryngitis, tracheitis, and bronchitis polyposa seu membranacea*. It not unfrequently extends over the whole of the air-passages, from the epiglottis to the minute ramifications of the bronchial tubes; and often affects the throat and pharynx, and sometimes even the œsophagus. It either attacks extensive continuous tracts of mucous membrane, or confines its ravages to isolated patches, and hence the exudation or croup-membrane either presents the appearance of continuous, tubular, arborescent coagula, corresponding with the division of the trachea and the bronchial ramifications, or of irregular patches, as is most commonly observed on the larynx. In bronchial croup, the tubular exudations from the larger bronchi present a calibre inversely proportional to their thickness, and those thrown off from the finer ramifications occur as solid cylinders.

The exudations present great differences in thickness and consistence; the membrane sometimes resembling an investment of hoar-frost, or gauze, whilst at other times it will even exceed a line in thickness, while the consistence may vary from that of viscid cream to that of the most compact, tough, leathery, coagulated fibrin. But neither the density nor the consistence is generally uniform throughout; the exudation, as a general rule, becomes thinner and gradually softer towards its edges, more puriform or creamy, and the portion in contact with the mucous membrane is the softer and looser of the two.

In colour they are yellowish white, or grey, and not unfrequently have a greenish tint; they either adhere firmly to the mucous membrane, or hang loosely on it, the latter being the case when a viscid secretion occurs between the false membrane and the mucous surface. The surface next to the mucous membrane is frequently marked with red streaks and dots, consisting in part of blood adhering to the surface, and in part, as found on closer examination, of straight or tortuous vessels, or of small, roundish extravasations, from which currents of blood are seen to emerge in an arborescent and radiating form.

The appearance of the subjacent mucous membrane is liable to considerable differences; its red colour sometimes assumes a very dark, almost brown tint, but more frequently a bright erysipelatous hue; and again it occasionally, but very rarely, happens that all signs of injection are absent; it presents an appearance of sores, as if it were excoriated, bleeds from numerous, minute, scattered spots, and presents various degrees of swelling. The swelling is, however, sometimes so very trifling as hardly to attract notice. The submucous areolar tissue is most commonly, if not always, the seat of serous infiltration.

Genuine croup of the air-passages is essentially a disease of childhood; it rarely, however, occurs before the end of the second year, and the parts it most commonly attacks are the larynx and trachea; in adults, bronchial croup is the most common variety, and during the age of puberty and early manhood, it is often associated with pneumonia. Croup of the final ramifications of the bronchi occurs simultaneously with pneumonia, and usually runs an acute course; sometimes, however, it assumes a chronic form, the process continuing with less intensity for a longer period, with occasional exacerbations, which give rise to the deposition of fresh products. In many persons it becomes habitual, and often, in the form of *bronchial croup*, seems to assume a certain degree of periodicity in its attacks. It is frequently combined with pneumonia, pleurisy, and pericarditis, and sometimes with meningitis, and acute and chronic hydrocephalus; and it occasionally extends to the stomach and degenerates into acute softening of that organ. It proves fatal from the contraction which it induces in the air-passages through exudation, and still more through the swelling of the mucous membrane over the subjacent areolar tissue, and from spasmodic closure of the glottis; moreover suffocation is frequently induced by *pulmonary œdema*, or the patient may occasionally sink from the exhaustion induced by very abundant exudation. We have no anatomical evidence that the pneumogastric nerve is seriously affected.

The other exudative processes yield a soft, purulent, and less plastic exudation, or a thin, sero-purulent, gelatinous, discoloured ichor, which attenuates, and finally dissolves the mucous membrane. The submucous areolar tissue is infiltrated

by a matter of a similar character, and its texture is rendered friable, lacerable, and fusible. These are, in most cases, secondary processes, depending on the localisation of a degenerated general disease of an acute exanthematous nature,—as variola or scarlatina.

All the exudative processes on the mucous membrane of the air-passages are frequently combined with similar processes on other mucous or serous membranes; they may degenerate into gangrene and acute softening; and from the development of the spleen, lymphatic glands, and follicular apparatus of the intestinal mucous membrane in these cases, we conjecture that they originate in a disease or dyscrasia of the whole mass of the lymph and blood.

Here we must also notice aphthæ of the air-passages; they are for the most part confined to the larynx, trachea, and the great bronchial trunks, very seldom extending to the throat; they scarcely ever occur as a primary affection, but are most commonly associated with tuberculous phthisis of the larynx and the lungs.

3. *Pustular Inflammation.*—The only form of pustular inflammation occurring in the air-passages is the *variolous*, which, however, is very perfectly developed. It is usually present whenever the variolous process exhibits considerable intensity, and when the skin is covered with an abundant eruption of the exanthema. It appears in the form of simple pustules on the mucous membrane of the epiglottis and adjacent soft palate, of the larynx, and the trachea, and not unfrequently in the bronchi and their primary branches. The pustules are soft, easily rubbed off, not unfrequently confluent, and when removed leave a superficial, concave, roundish spot, where the mucous membrane presents a dark red or livid tint, and an appearance of excoriation. Between these spots it presents various degrees of redness and thickening, and is coated with a tough plastic mucus,—a croupous exudation; moreover it is much swollen, and together with the submucous areolar tissue, exhibits signs of serous infiltration. Very intense confluent pustulation may give rise to *variolous ulceration*.

4. *The Typhous Process on the Mucous Membrane of the Air-Passages.*—The typhous process occurring in the air-passages presents numerous peculiarities in reference to its connection

with the general disease, with the morbid state of the mucous membrane of the small intestine, where amongst us it usually becomes localised as ileo-typhus, and in reference to its seat generally.

In all cases of typhus it invariably occurs as a *typhous bronchial catarrh*, with tough and gelatinous-looking mucus. The catarrh seems to be developed in proportion to the intensity of the general disease, and is most severe in those cases which are marked by the predominance of catarrho-pectoral symptoms. It may occur here as the *true, special typhous process*, either in its genuine or its degenerate form; in this former case it may be either *primary* or *secondary*; in the latter case it is *always secondary*. Its seat is sometimes on the bronchial and at other times on the laryngeal mucous membrane; on the former it frequently occurs as *primary broncho-typhus*, and is a very serious affection; on the latter, constituting *laryngo-typhus*, it is almost always, at least amongst us, a secondary process.

A. *Genuine typhus on the bronchial mucous membrane* always appears as an intense, diffused congestion; the mucous membrane is of a dark, almost violet tint, is swollen and succulent, and yields a secretion of a gelatinous and sometimes dark, blood-streaked mucus, occurring in large masses. The disease is most commonly developed in the bronchial ramifications of the lower lobes; it is always limited to the stage of typhous congestion, and never gives rise to any apparent production of a secondary formation on the tissue of this membrane, such as is produced in immense quantity in the intestinal follicles in cases of abdominal typhus.

In *primary broncho-typhus* the general disease originally localises itself here, avoiding all other mucous membranes, even that of the intestine, for which the typhous process in general shows the most decided preference; the latter mucous membrane exhibits, however, in many cases a recognisable, although always subordinate and secondary development of the follicles, in which the adjacent mesenteric glands participate; and in such cases it is very often a difficult matter to distinguish the typhus in the above-named affection of the bronchial mucous membrane. The peculiar stasis of the spleen and of the great *cul de sac* of the stomach, the remarkable intumescence of the

former, and the singular character of the blood, the typhous nature of the general disease, and especially the altered condition of the bronchial glands, invariably serve, together with other symptoms, to indicate the typhous nature of the bronchial affection. The alteration occurring in the bronchial glands is of the same character as that affecting the mesenteric glands in abdominal typhus; they become swollen to the size of a pigeon's or even a hen's egg, are of a dark, violet colour, which afterwards becomes lighter, present a relaxed and friable appearance, and are infiltrated with medullary typhous matter. Like typhous mesenteric glands they may become the seat of a tumultuous metamorphosis, and thus, either with or without perforation of the adjacent mediastinum, may give rise to pleurisy.

This form is often combined with pneumo-typhus and typhous pleurisy, and is beyond all doubt the basis of the spotted contagious typhus, and very probably, also, of the Irish and North American typhus, which, in the majority of cases, run their course without any intestinal affection. With us this affection is rare, and, in point of frequency, is not to be compared to abdominal typhus.

Genuine secondary bronchial typhus presents the same anatomical characters, in a less highly developed state, as the primary. In a *degenerate* form it is very rare, occurring, for the most part, as bronchial croup, or as diffuse gangrene of the bronchial mucous membrane.

B. *Laryngo-typhus* is with us an unusually common and extremely unfavorable symptom in many epidemics of typhus. It scarcely ever occurs as a primary independent affection, but is almost invariably secondary, and forms, as it were, the completion of intestinal typhus, on various anomalies of which it is generally based.

It is almost invariably situated on the laryngeal mucous membrane above the transverse muscle, and towards the posterior extremities of the ventricles (a situation which, as we shall presently see, appears favorable to all pseudo-plastic processes); it may, however, occur on the mucous membrane of the epiglottis, especially towards its lateral borders; and sometimes it occurs simultaneously at both these spots.

It, no doubt, frequently occurs in the *genuine* form, but it

is only rarely that we have the opportunity of observing the typhous infiltration in its stage of crudity or of metamorphosis; as we see it in the dead body, there is almost invariably a loss of tissue, or ulcers of the same kind as those in the intestine, but less deep-seated.

Laryngo-typhus occurs, however, far more frequently in a *degenerate* form, either as an *exudative process* (croup), or more commonly as *gangrene*. The latter, after its detachment, leaves an ulcer, which cannot be distinguished from the degenerate typhous ulcer, so that we are unable from these appearances to draw any certain inference regarding the original process.

These ulcers are of a roundish shape, varying from the size of a lentil to that of a pea; they are either discrete or confluent, two or three often forming a group. They are seated at the spots we have already mentioned, on the posterior wall of the larynx and on the lateral edges of the epiglottis, on both of which situations they occur as linear ulcers; when, as is sometimes the case, they present themselves on the inferior surface of the epiglottis, they present a roundish or lenticular form; they are lax, discoloured, and are black at the edges from the deposition of pigment; they gradually eat their way into the transverse muscle, the arytenoid and cricoid cartilages, the vocal chords and epiglottis, in which they give rise to softening, necrosis, and exfoliation. On the posterior laryngeal wall abscesses are not unfrequently developed, in which the necrosed arytenoid cartilages lie bathed in a brownish ichor; these abscesses sometimes penetrate into the pharynx. The whole constitutes a *typhous laryngeal phthisis*.

Laryngo-typhus is very frequently combined with pneumonia, and with secondary broncho- and pharyngo-typhus.

c. Inflammation of the Submucous Areolar Tissue.—In addition to the part that the submucous areolar tissue takes in inflammation of the mucous membrane of the air-passages, it is also subject to inflammations, occurring as primary affections. These inflammations are, however, rare, and for the most part of a metastatic character; and hence they have a special tendency to run into suppuration and necrosis of the areolar tissue and mucous membrane. In reference to their position and diffusion over the air-passages,

they are usually limited to the submucous areolar tissue of the *larynx*; they may, however, extend to the corresponding tissue in the throat and pharynx, and even into the intermuscular areolar tissue of the neck. As results of *chronic* inflammation, we often, in the larynx, meet with *hypertrophy*, *thickening* and *callous induration* of this tissue, and a consequent *narrowing of the cavity of the larynx*.

d. Ulcerous Processes.—In the course of the preceding observations we have already noticed some of these processes; others still require to be described. Their position is, with very few exceptions, in the larynx and trachea, and as they are generally the result of a process originally proceeding from the mucous membrane, the direction of their destroying course is inwards into the tissues, and, as a rule, they make their way from within outwards.

Those we have already described are *catarrhal suppuration*, the *sloughing ulcer*, which, with the *aphthous ulcer*, must be regarded as degenerate exudative processes, the *variolous ulcer*, and the *typhous ulcer*; also the *suppuration and necrosis of the mucous membrane, proceeding from the submucous areolar tissue*. We have yet to consider *suppuration of the perichondrium with necrosis of the laryngeal cartilages*, and *tuberculous and cancerous ulceration*.

We must here notice syphilitic ulceration of the air-passages. Ulcers of this nature are, for the most part, situated on the epiglottis, having extended there from the soft palate and the root of the tongue. They usually present the characters of secondary chancres, and not unfrequently give rise to entire destruction of the epiglottis and of the mucous membrane around the glottis. The syphilitic destruction usually confines itself to these parts, leaving, after cure, a loss of part of the epiglottis, and thick, hard, white, tendinous, and cord-like cicatrices crossing one another and giving rise to contraction. In rare cases, however, they extend to the larynx and trachea, and destroy the mucous membrane by causing sloughing and aphthæ, giving rise to contraction of the submucous tissue, and to friability and brittleness of the cartilages.

The walls of the air-passages are also liable to ulceration from without inwards. This occurs by far the most frequently in

the bronchi, in consequence of their frequent proximity to softened tubercle and to tuberculous abscesses.

e. Edema of the Mucous Membrane of the Air-Passages.—This affection has especially attracted the attention of pathologists when it has been situated in the larynx, where it has received the name of *œdema glottidis*. It is in this position that its attacks are most intense, and that its consequences are the most dangerous. In some few cases it extends to the mucous membrane of the posterior walls of the trachea and pharynx.

In the cases strictly falling under this head, it occurs as an infiltration of the submucous areolar tissue and of the mucous membrane itself, with a colourless or pale-yellow serum. When it occurs as *œdema glottidis* it is situated in the mucous membrane of the epiglottis, the duplicaturæ aryepiglotticæ, and the mucous membrane of the vocal chords and ventricles; and it constitutes a transparent pale-yellow, fluctuating tumour, which, in proportion to its size and extent, diminishes the aperture of the glottis, and may even entirely close it.

Œdema of the glottis, either in an *acute* or *chronic* form, may accompany not only all the inflammatory processes of the laryngeal mucous membrane of which we have spoken, but many other morbid conditions of the larynx and adjacent parts connected either essentially or incidentally with an irritation of the aforesaid mucous membrane. It accompanies catarrhal inflammations, especially those of an exudative nature, exanthematous processes, typhous and all ulcerous processes on the laryngeal mucous membrane, inflammations of the submucous tissue, tuberculous or cancerous affections of the larynx, &c.

These cases are of the highest importance, for the affection may become *rapidly* developed, and may cause death by asphyxia in any of the above-named affections of the laryngeal or adjacent mucous membrane, as that of the velum palati or tonsils, even when the primary disease seems trifling; and it unfortunately happens that we are entirely ignorant of the peculiar conditions under which it is produced in these cases. Seropurulent infiltration of the submucous areolar tissue may be confounded with true *œdema*; the former is, however, invariably the result of an intense inflammatory process.

f. Gangrene of the Air-Passages.—This affection occurs both here and in the parenchyma of the lungs in two distinct forms, either as a *circumscribed eschar* on the mucous membrane, eating its way into the submucous tissue, in which it may also occur primarily, or as a *diffuse gangrenous colliquescence of the bronchial mucous membrane*. The conditions under which it is developed are similar to those of gangrene of the lung, with which it is sometimes combined. It generally, however, occurs in tissues in some way previously diseased, but appears rather as an accidental termination than as a necessary consequence of any peculiar local morbid process. We have seen it take its origin from inflammation of the perichondrium of the laryngeal cartilages, from tuberculous laryngeal phthisis, from typhous ulcers, and laryngeal croup, and give rise to *circumscribed gangrenous destruction*, or much more frequently to *diffuse gangrene of the bronchial mucous membrane*. In the latter case, we find a certain extent of the mucous membrane either uniformly or at certain spots, of a dirty brownish-green colour, and broken up into a soft, villous, moist, friable tissue, evolving the peculiar odour of sphacelus. The tubes are filled with a corresponding, discoloured, frothy, stinking, sero-ichorous fluid. It is most commonly associated with pulmonary gangrene.

g. Adventitious Products.—See the remarks at *d*, page 30.

B. Diseases of the Cartilaginous Skeleton of the Air-Passages.

a. Inflammation of the Perichondrium of the Laryngeal Cartilages. (Perichondritis laryngea.)—In the examination of the dead body we have occasional opportunities of noticing a peculiar form of suppuration in the larynx, undoubtedly resulting from inflammation that had commenced in the perichondrium, which appears to be detached either at circumscribed spots, or more commonly over both surfaces of a whole cartilage, and under this a quantity of pus is found collected in a membranous sac. The cartilages are more or less denuded, rough, villous, necrosed, and perforated, or they lie entirely free, discoloured, attenuated, softened, and more or less disintegrated in a large collection of pus. This abscess may make its way into the

larynx, trachea, or pharynx, or may even open and discharge its contents externally.

This disease seems most frequently to attack the cricoid cartilage; it is commonly supposed to be of a rheumatic origin, and has been termed *rheumatic laryngeal phthisis*; it may, however, also occur as a consequence of the acute exanthemata (at least of variola) and of the mercurial disease.

b. Inflammation and Softening of the Epiglottis.—The epiglottis is sometimes the seat of a chronic inflammatory process, which finally leads to its conversion into a dense, rigid fibro-cartilaginous tissue,—a change attended by shrivelling and deformity.

As a contrast to this rigidity, we may have softening of the epiglottis. This is probably also a result of inflammation, and is similar to the softening that occurs in the yellow coat of the arteries. The epiglottis loses its elasticity, becomes soft and friable, assumes a dirty yellow tint, and at length begins to waste away.

c. Ossification.—In the more advanced period of adult life, the cartilages of the larynx in the male are always more or less ossified; hence we need only notice those cases in which this change commences in early life at a more than ordinarily rapid course, or in which it spreads over an unusually great extent. The following is the order, in regard to frequency, of the parts thus morbidly affected; the thyroid cartilage, the cricoid cartilage, the tracheal rings, and the bronchial cartilages; it is extremely seldom that the arytenoid cartilages are affected. The ossification is here a true conversion of the cartilage into actual bone. The change may either occur spontaneously, or result from an inflammatory vascular activity in the perichondrium and cartilage, as is evidenced by its frequent occurrence at and below the seats of ulcers, especially those of a tuberculous character. Moreover, fractures and injuries of the cartilage give rise to this affection by inducing a deposition of ossifying callus. The newly-formed bone, in cases of laryngeal phthisis, may become the seat of caries and necrosis. Fragments of bone may then be expectorated, which, by the peculiar characters of their tissue, may be distinguished from various other earthy concretions that are sometimes ejected by coughing.

In certain rare cases we find the ossification extending not

only to the most minute bronchial cartilages, but even to the finest of the membranous bronchial twigs. A system of rigid arborescent tubes then pervades the lungs, and hinders their collapse if a section be made through them; on passing the finger over the cut surface, a sensation is perceived similar to that produced by projecting, angular grains of sand. This change only occurs in very aged persons.

The *epiglottis* is never, strictly speaking, ossified, but we occasionally find that, when its texture has been modified by inflammation and has assumed a fibroid character, its form becomes variously modified, and it contains earthy deposits.

d. Adventitious Products.—Adventitious formations occurring in the air-passages are of the highest importance when they project into the interior of the air-passages in the form of broad or pedicled vegetations, and thus give rise to more or less contraction of their calibre. They occur almost exclusively in the larynx, and they are classified and treated of, according to their external characters, under the general head of laryngeal tumours. When considered in reference to their internal structure, they may be reduced to the following forms, which may be developed in and under all mucous membranes.

1. *Epithelial Formations.*—These occur in the form of roundish cauliflower or wart-like growths, varying from the size of a hemp-seed to that of a hazel-nut, and are occasionally even larger; they are attached by a short pedicle to the mucous membrane, present a somewhat lobular and laminated structure, and consist of exuberant epithelial cells and very delicate vessels, prolonged, as it were, from the mucous membrane. They are especially liable to occur on the vocal chords and arytenoid cartilages, but they are sometimes found on the under surface of the epiglottis, and on the cricoid cartilage. As they frequently spring from a cancerous basis, they are often of a malignant nature, but they have also been observed in a non-malignant form in persons of various ages after catarrhs and repeated attacks of croup. They are the most common of all laryngeal tumours.

2. *Cellular or Mucous Polypi and Condylomatous Excrescences.*—These occur upon or in immediate proximity with an ulce-

rated basis, or when there are no existing ulcerations, they form hard or spongy purple vegetations varying from the size of a pin's head to that of a hempseed or pea, lying sometimes in great numbers closely beside one another, and occupying large portions of the laryngeal mucous membrane. Their favorite seat is the mucous membrane of the vocal chords. They are most probably of a syphilitic nature, and when, as is sometimes the case, they are associated with tuberculous laryngeal phthisis, and constitute the tuberculous ulcer, we are led to suspect that they originate in a combination of the tuberculous with the syphilitic dyscrasia.

3. *Erectile Tissue* occurs in the form of broad-based, soft vegetations, capable of being rendered turgid, or as the development of the free extremities of mucous polypi. The former variety not unfrequently springs from a cancerous basis.

4. *Fibrous tumours*.—These are extremely rare in the submucous areolar tissue of the mucous membrane lining the cavity of the larynx, but are more common beneath the pharyngeal mucous membrane investing the posterior wall of the larynx. They are here often found not only of their ordinary inconsiderable size, but sometimes of a very large volume, and by their adhesion to the pericardium, remind us of the large pharyngeal polypi springing from the submucous periosteum.

5. *Cancer*.—We have already remarked that cancer not unfrequently forms the basis of exuberant epithelial formations and erectile tissues. It further occurs in the larynx as fibrous cancer, in the submucous areolar tissue as medullary cancer, and (which is extremely singular) as cancerous degeneration of the *arytenoid cartilages*. According to the volume of the morbid product, there are found larger or smaller nodular roundish protuberances into the laryngeal cavity, which thus becomes more or less diminished in size. These cancerous tumours, for the most part, prove fatal while still in their state of crudity; they sometimes, however, undergo their ordinary course of metamorphosis, and give rise to a cancerous ulcer.

Cancer also occurs in the trachea and bronchi, but in such cases it is almost invariably only a secondary affection. Thus it occasionally happens that the trachea is perforated by sur-

rounding masses of medullary cancer in the neck; more frequently, in which case the bronchi may also be affected, by cancerous accumulations in the mediastinum posticum, or by cancer of the œsophagus; and in these cases the air-passages become contracted by the growth of vegetations within them. In the bronchi we sometimes observe an ordinary cancerous degeneration of the fibrous sheath, proceeding in different directions from a bronchial stem along its ramifications, by which their walls are thickened and rendered rigid, and their calibre is diminished, while their inner surface becomes nodular and uneven. This degeneration appears to arise from cancerous disease of one or more of the bronchial glands.

6. *Tuberculosis of the Air-Passages*.—Tubercle is very commonly met with in the air-passages, but it is found in some parts of them much more frequently than in others. The most common position is in the larynx; it is very rare in the trachea and larger bronchi; while, again, it is not uncommon in the ultimate ramifications of the tubes. On softening it gives rise to tuberculous ulceration, and to laryngeal, tracheal, or bronchial phthisis, according to the seat of the deposit.

Tuberculosis of the larynx, as a primary and independent affection, is so extremely rare that we feel inclined to doubt its existence. It is almost invariably developed as a consequence of pulmonary tuberculosis, and then, as a general rule, not until the affection has established itself as pulmonary phthisis, and made considerable progress. The seat of tubercle is almost constantly and exclusively the mucous membrane and submucous areolar tissue lying over the transverse muscle and the adjacent arytenoid cartilages; it occurs, however, exceptionally at other spots, as for instance, the anterior surface of the epiglottis. It is either deposited in the form of grey granulations in the submucous areolar tissue, or as yellow, caseous, friable, tuberculous matter, is infiltrated into the mucous membrane; in either case, however, and especially in the latter, it rapidly softens, and ulceration is established. The softened, grey granulations form small roundish ulcers, varying from the size of a millet-seed to that of a lentil, with raised, hard edges. These unite with one another, and give rise to a secondary form of ulcer, irregular in shape, with pouch-

like indented edges, and a cellular, callous, thickened base, both of which may become the seat of secondary tuberculous deposit. The tuberculous infiltration becomes disintegrated in the mucous membrane, and forms therewith an extremely irregular and, as it were, gnawed and fissured ulceration, presenting obvious signs of reaction, namely, redness, injection, swelling, œdema of the tissues, and aphthous exudation over the adjacent parts.

The ulcer enlarges in consequence of secondary tuberculous deposition at its edges and the surrounding parts, as well as on its base, superficially as well as deeply; it thus gives rise to ulcerations which extend over the whole larynx and epiglottis, upwards to the soft palate and root of the tongue, downwards to the trachea, and inwards, causing suppuration and necrosis of the fibrous tissues and of the cartilages. They may even perforate the larynx from within outwards, and give rise to emphysema.

The secondary tuberculous ulcer is sometimes distinguishable by a condylomatous development of the mucous membrane at its edges, and of the islets of mucous membrane, which, as the ulcer enlarges, are frequently left on its base. It is not improbable that, in these cases, the tuberculosis is combined with syphilis.

There can be no doubt that in some rare cases, and under the requisite general conditions, tuberculous ulcers of the larynx are healed; but they always leave an unshapely cicatrix, puckered in proportion to the extent of the ulcer, and callous in proportion to its depth. We must, however, be careful not to regard all the cicatrices which we may find in the neighbourhood of true tuberculous ulcers in the larynx and trachea, as the cicatrices of so many tuberculous ulcers.

Tuberculosis of the trachea is extremely rare, it being only seldom that the corresponding laryngeal affection extends itself in a tuberculous ulcer to the upper part of the canal. In laryngeal phthisis we often, however, meet with small ulcers on the tracheal mucous membrane, and frequently in such numbers that they present a confluent appearance. These are the ulcers to whose cicatrices we referred, as liable to be mistaken for the cicatrices of tuberculous ulcers. They are small, shallow ulcers, most commonly of an oval but sometimes of a linear

form, with a very slightly concave base; the depression being so trifling that it is sometimes only detectible when the light falls obliquely on it: this base presents a raw and excoriated appearance, is of a pale or dark red colour, and is either exposed or coated with a creamy, diffuent exudation of a croupous nature; and it is surrounded by a fiery redness, or by a sharply defined red areola. They are most commonly situated on the posterior wall of the trachea, and frequently extend into the bronchial trunks, and we often find them much more numerous on one side (that, namely, in which the most diseased lung is situate) than on the other; moreover they are found with tolerable frequency in the pharynx and on the mucous membrane lining the mouth. They present nothing in common with tuberculous ulcers, and consist, as is at once seen, in an exudative aphthous process which is frequently associated with florid, laryngeal phthisis. If the tuberculosis be in a state of arrest, or actually retrograding, the above-described erosions become replaced by delicate, whitish, glistening, radiating, or star-like cicatrices.

Bronchial Tuberculosis.—This is seated in the bronchial mucous membrane, which becomes so infiltrated with yellow, lardaceous, caseous, tuberculous matter, as finally to appear converted into it. The bronchial tube itself becomes considerably enlarged, its calibre becoming at length completely obstructed by tuberculous matter, while its fibrous sheath becomes infiltrated with lardaceous matter, callous, and thickened. This degeneration sometimes attacks the bronchial mucous membrane as a *secondary* affection, in which case it arises from tuberculous abscesses, and affects the tubes opening into them; it is then primarily dependent on pulmonary phthisis.

Primary bronchial tuberculosis is a much more important affection. It is, as we have already mentioned, a disease of the ultimate ramifications of the bronchial tubes, arising originally in them, and extending backwards to the larger bronchi. Like pulmonary tuberculosis, it most commonly occurs in the bronchial ramifications of the upper lobes, but it stands contrasted with that affection in frequently occurring in the peripheral branches; it attacks a larger portion of the bronchial tree, and on making a section of the pulmonary parenchyma we

find it traversed by thick-walled, dilated, bronchial tubes filled with caseous tuberculous matter.

Bronchial tuberculosis, although very frequently combined with lardaceo-gelatinous, or fatty and caseous tuberculous infiltration of the lungs, sometimes occurs as an independent disease. In the latter case the obstruction in the bronchial tubes gives rise to obliteration of the pulmonary vesicles and obsolescence of the parenchyma connected with them; and, on making an incision, we then find the obstructed tuberculous bronchi ramifying through the parenchyma in the form of ribands of puckered, tough, elastic tissue.

The tuberculous matter may undergo either of the two following changes. It may soften; and in this way it not unfrequently destroys the bronchial walls, and gives rise to tuberculous abscesses in the adjacent parenchyma. The abscesses arising primarily from the destruction of the bronchus are incomparably rarer than those arising from the softening of pulmonary tubercles. This metamorphosis is most likely to occur when tuberculous infiltration of the parenchyma is simultaneously present. The other change to which we referred is the cretification of the tuberculous matter. This metamorphosis most commonly occurs when the bronchial tube has been completely obstructed by tuberculous matter, and the pulmonary tissue to which it pertains has become obsolete; under favorable conditions it seems, however, sometimes to occur in other cases, in which the morbid product becomes as it were disintegrated into a caseous pultaceous mass, which, instead of becoming softer, thickens and becomes ultimately converted into a chalky substance, around which the bronchial tube becomes contracted and atrophied.

Bronchial tuberculosis, as a primary affection, is most common in childhood, and is usually associated with all the tuberculoses of other organs peculiar to this period of life, and especially with intense *tuberculosis of the bronchial glands*.

Its most marked analogies are exhibited in tuberculoses of the mucous membrane of the Fallopian tubes and uterus.

7. *Anomalies of the Contents of the Air-Passages*.—We must here notice:

1. The products of various morbid processes on the mucous membrane of any portion of the air-passages, such as blood,

or as mucus, (which may collect in large quantity, and may present many peculiarities of character, being grey or pearl-coloured, colourless, transparent, aqueous, viscid, glassy, of a creamy, whitish-yellow colour, or puriform,) or as true pus, or membranous exudations (croup-membranes), or as ichorous fluid, tubercle and tuberculous pus, fragments of necrosed cartilage, ossified bronchial cartilage, &c.

2. Products of morbid processes, occurring external to the air-passages, and finding their way into them, either by the natural passages, or by a destruction of tissue,—as for instance *blood*, (usually in considerable quantity,) in a coagulated or fluid state from the lungs or from aneurisms which have opened into the air-passages; *serous frothy fluid* (bronchial froth) from the parenchyma of the lungs; *pus and ichor* arising most frequently from abscesses in the lungs and bronchial glands or in the vertebræ; the corrosion of a bronchial trunk may allow the fluid of empyema to be discharged into the air-passages, and by another process the contents of an hepatic abscess may make their way into the bronchial tubes; the *ichor of cancer* principally arising from cancerous destruction of the œsophagus; *masses of tubercle*; *calcareous* and *stony concretions*, with which, as produced in the air-passages, we must place cretified blenorrhœal mucus and tubercle; and, finally, *acephalocysts* from the lungs, liver, and thyroid gland (Portal).

3. Foreign bodies which—

(a.) Find their way from the pharynx and œsophagus, or even from the stomach and intestinal canal, by means of abnormal modes of communication, or by the natural passages, into the air-tubes. The most common are fluids that have been imbibed, and having made their way through ulcerous or cicatrised strictures, penetrate into the trachea or bronchi. Amongst the foreign bodies proceeding from the stomach and intestinal canal, we must especially notice the round worm, which has been seen both by ancient and modern observers in the pharynx in children, and, crawling into the glottis, has produced death by suffocation.

(b.) Articles of food may become impacted in the glottis, in cases in which the act of deglutition is impeded by inflammation and degeneration of the muscular walls of the pharynx, or by enlarged tonsils, or is inattentively performed from haste

and carelessness as in imbeciles, or is interrupted by coughing, sneezing, or laughing. These accidents are most liable to occur when, from atrophy, rigidity, or malformation, the epiglottis is no longer able sufficiently to protect the glottis; and the substances most commonly causing them are large pieces of tough meat, skin, and gristle.

(c.) Foreign bodies which accidentally enter the glottis independently of any intentional act of swelling, and either fall into the trachea and bronchi, or are forced into them by an automatic movement of deglutition. Cases are recorded in which the following bodies have got in the air-passages:—small arrows, plum and cherry stones, small coins, natural and artificial teeth, ears of corn, nails, pebbles, and fragments of glass. In favorable cases they are soon removed by coughing; but otherwise they remain for a long time in the air-passages, inducing not only inflammation of the mucous membrane, pneumonia, and ultimately suppuration, but even destruction of the walls of the bronchial tubes. This they may effect in various ways, and they may even penetrate into the adjacent blood-vessels. I may here mention the following singular case: A little boy sucked a dart from a blowing-tube. Its feathered portion was downwards as it descended the trachea, and from thence it went into the left bronchus. On the twelfth day he died from hæmorrhage of the air-passages, having at the same time symptoms of pneumonia. Dissection revealed bronchitis, especially of the left side, and hepatisation of the left lower lobe. The dart was lying loose in the left bronchus, with its feathered part downwards. Opposite to the opening of this bronchus into the trachea, in the cartilaginous wall of its right side, there was an injured spot of about the size of a hemp-seed, and through this there was a perforation into the adjacent *arteria innominata*. In the paroxysms of coughing, the point of the dart was being constantly forced against this spot in the right tracheal wall, which lay opposite to the axis of the left bronchus; and in this way the fatal lesion was ultimately produced.

It is neither unimportant nor uninteresting to remark that, in the majority of cases, these foreign bodies fall into the right bronchus (Key). This is undoubtedly dependent on its larger size, on the greater obtuseness of the angle that it makes with

the trachea, and on the greater energy of the current of air rushing through it. Moreover, this is in accordance with the well-known fact that in new-born children respiration is effected sooner and more perfectly by the right bronchus and lung than by the left.

II.—ABNORMAL CONDITIONS OF THE PLEURA.

§ 1. *Deficiency and Excess of Formation.*

The pleural sacs are altogether absent when there is an entire deficiency of the respiratory apparatus, as in cases of acephalia, the thoracic cavity being then filled with a dense fibro-cellular tissue. A partial deficiency occurs when, in consequence of the absence of the diaphragm, the pleura and peritoneum form one continuous membrane.

An excessive development occurs in the form of *duplication* of the pleura, except in cases of double monstrosity, when the thoracic cavity is more or less doubled, or the two lungs are deposited in a large common cavity. It is, however, extremely rare, especially as contrasted with the frequent occurrence of congenital duplications of the peritoneum; and on this account, as well as in consequence of its relations, I would direct attention to a hitherto unnoticed persistent duplication of the *right* pleural sac. It forms, at the obtuse apex of the pleura, a fold hanging from above downwards, and from without inwards, including the arch of the vena azygos, and lying in a supernumerary fissure, which divides the upper lobe into two parts.

§ 2. *Anomalies in the Size and Form of the Pleural Sacs.*

The size and volume of the pleural sacs are proportional to the congenital or acquired volume of the lungs; thus, for large lungs there are wide and spacious pleuræ, while for small lungs the pleural sacs are less capacious. When the lungs become enlarged, as in emphysema, they also enlarge; when the lungs, from any morbid process, become atrophied, they also diminish in a corresponding degree. Moreover the pleural cavities may undergo various degrees of enlargement by the accumulation within them of gases or liquids, and may be diminished in various ways by an increased size of the peritoneum or

pericardium, by adventitious products in the mediastina, or by malformation of the bony case of the thorax.

The form of the pleural sacs is regulated by that of the osseous thorax, and we must refer our readers to the remarks on the anomalies in the form of that cavity.

§ 3. *Diseases of Texture.*

a. Hyperæmia of the Pleura.—A continuous increased flow of blood to this membrane gives rise, according to its extent, either to local or general opacity, and to hypertrophy; and ultimately to the development of an anomalous cartilaginous and osseous substance in its texture, and in that of the subjacent areolar tissue.

On the other hand, congestion may cause increased secretion, which, in accordance with its character and the general state of the blood, may terminate in a temporary or permanent accumulation of various quantities and kinds of serous fluid, constituting hydropspleuræ or hydrothorax. It is very seldom that it leads to hæmorrhage of the pleura or hæmothorax.

b. Inflammation of the Pleura (Pleuritis, Pleurisia).—This is the most frequent of the diseases of the pleura; it generally appears as an idiopathic and primary affection, most commonly of a rheumatic nature; in consequence of a wound or shock affecting the walls of the thorax, or of the contact of the pleura with the atmospheric air, either from without or through the air-passages from within, or of the contact of pus, ichor, &c.; or it may originate in the extension of inflammation or other morbid processes in neighbouring parts, and especially the lungs; and it very often is a secondary or metastatic process, and is then frequently associated with inflammation of other serous membranes, especially of the peritoneum and pericardium; it generally exhibits a very well-marked croupous character.

Pleurisy is either *general* or *partial and circumscribed*; in the former case the process on the visceral surface (the pulmonary pleura) as a general rule exhibits comparatively little intensity. In either case the disease may be *acute* or *chronic*. As everything that has been stated with respect to inflammations of serous membranes generally applies to inflammations of the pleura, we shall here limit our remarks to the notice of certain important peculiarities presented by this membrane.

The exudations present all the differences that we have there described; but here we must especially notice, as frequent and very important forms, the *purulent* exudation (empyema) and the *hæmorrhagic*. In relation to the quantity of the exudation, acute, and more especially chronic pleurisies depositing a long continuing and paroxysmally increasing exudation in large quantity, are of the greatest importance. When the pleurisy is general, the exudation sometimes amounts to eight, ten, sixteen, or twenty pounds, and occasionally even more. The walls of the thorax become in these cases more or less dilated; the intercostal spaces are enlarged, and in consequence of the paralysed state of their muscles, are flattened; the diaphragm is forced downwards in the abdomen; the mediastinum and the heart are pushed to the opposite side, and thus diminish its capacity. The lung itself is compressed by the effusion, in a degree corresponding to its quantity, and if no old adhesion exists to oppose it, it is constantly pressed upwards and inwards on the mediastinum and the vertebral column. We find it thus compressed to the fourth, sixth, or even the eighth part of its normal volume, and so flattened on its external arched surface as to present the appearance of a flat cake; its texture is of a pale red, bluish brown, or lead-grey tint; and it is of a leathery toughness, and devoid of blood and air. In this state its external surface is invested with a plastic coagulum; as this extends over it to the costal pleura, the lung is, strictly speaking, excluded from the cavity of the sac formed by the pleuritic exudation. If adhesions already exist, as the remains and consequences of previous inflammations, they will, in proportion to their position, distribution, the tissues comprising them, and their powers of resistance, present a certain amount of opposition, as we have already described, to the displacement; and the degree of dislocation of the lung must be thereby more or less modified. In partial pleurisy the displacement and compression are limited to the portion of the lung corresponding to the extent of the affection.

The *purulent* exudation most commonly accompanies the pleuritic process in weak, cachectic persons, whose organisms seem prone to form pus; on the other hand, it may, by an intensely high degree of inflammation, and by its frequent recrudescence, give rise to very rapid general debility, cachexia,

and pyæmia. The effused pus not unfrequently degenerates into ichor, and this change is sometimes accompanied by the development of gas, so that from its decomposition and disintegration a pneumothorax becomes added to the purulent effusion. This not unfrequently leads to suppuration of the walls of the chest, with or without caries, and to the spontaneous discharge of the collected fluid, or to suppuration of the lung from its surface, leading to perforation of the bronchial tubes, or to suppuration of the principal bronchial trunks, whereby, on the one hand, atmospheric air is allowed to penetrate the pleural sac, and, on the other, pus enters and is discharged through the bronchi.

Amongst *partial* pleurisies we must especially notice those which occur about the apices of the lungs (for the most part dependent on pulmonary tuberculosis), those in the inferior portion of the pleural sac and on the pleura diaphragmatica, those on the laminæ of the mediastinum, and finally those which occur in the interlobular fissures of the lungs.

Plastic exudations, when they result either from an acute or a chronic simple process, (by which we mean to imply a process not implicating the exudation,) become transformed into areolar tissue of various degrees of density, into areolo-serous and fibrous tissues, presenting varied relations in reference to form and distribution.

When they are the products of general pleurisy they invest the whole of the costal and pulmonary pleura; or they merely occur at individual spots in the form of circumscribed patches on the serous membrane.

The areolar tissue forms either dense and rigid bands, or thready, lax, and moveable adhesions, according to the comparative absence or presence of aqueous effusions, occurring either originally or during its formation. These adhesions are of very common occurrence; when the lung adheres at every point to the parietal surface of the pleura, they are termed *general*, and when the latter is only attached to a portion of the lung, *partial* (or *cellular*) *adhesions*. They may undoubtedly become the seat of new inflammatory processes, but, as Laennec remarked, they usually limit the progress of new pleurisies. When general dropsy and the dropsical diathesis are present, they may become the seat of a serous, briny infiltration.

The exudations present all the differences that we have there described; but here we must especially notice, as frequent and very important forms, the *purulent* exudation (empyema) and the *hemorrhagic*. In relation to the quantity of the exudation, acute, and more especially chronic pleurisies depositing a long continuing and paroxysmally increasing exudation in large quantity, are of the greatest importance. When the pleurisy is general, the exudation sometimes amounts to eight, ten, sixteen, or twenty pounds, and occasionally even more. The walls of the thorax become in these cases more or less dilated; the intercostal spaces are enlarged, and in consequence of the paralysed state of their muscles, are flattened; the diaphragm is forced downwards in the abdomen; the mediastinum and the heart are pushed to the opposite side, and thus diminish its capacity. The lung itself is compressed by the effusion, in a degree corresponding to its quantity, and if no old adhesion exists to oppose it, it is constantly pressed upwards and inwards on the mediastinum and the vertebral column. We find it thus compressed to the fourth, sixth, or even the eighth part of its normal volume, and so flattened on its external arched surface as to present the appearance of a flat cake; its texture is of a pale red, bluish brown, or lead-grey tint; and it is of a leathery toughness, and devoid of blood and air. In this state its external surface is invested with a plastic coagulum; as this extends over it to the costal pleura, the lung is, strictly speaking, excluded from the cavity of the sac formed by the pleuritic exudation. If adhesions already exist, as the remains and consequences of previous inflammations, they will, in proportion to their position, distribution, the tissues comprising them, and their powers of resistance, present a certain amount of opposition, as we have already described, to the displacement; and the degree of dislocation of the lung must be thereby more or less modified. In partial pleurisy the displacement and compression are limited to the portion of the lung corresponding to the extent of the affection.

The *purulent* exudation most commonly accompanies the *suppurative* process in weak, cachectic persons, whose organisms are unable to form pus; on the other hand, it may, by an intense high degree of inflammation, and by its frequent recurrence, give rise to very rapid general debility, cachexia,

and pyæmia. The effused pus not unfrequently degenerates into ichor, and this change is sometimes accompanied by the development of gas, so that from its decomposition and disintegration a pneumothorax becomes added to the purulent effusion. This not unfrequently leads to suppuration of the walls of the chest, with or without caries, and to the spontaneous discharge of the collected fluid, or to suppuration of the lung from its surface, leading to perforation of the bronchial tubes, or to suppuration of the principal bronchial trunks, whereby, on the one hand, atmospheric air is allowed to penetrate the pleural sac, and, on the other, pus enters and is discharged through the bronchi.

Amongst *partial* pleurisies we must especially notice those which occur about the apices of the lungs (for the most part dependent on pulmonary tuberculosis), those in the inferior portion of the pleural sac and on the pleura diaphragmatica, those on the laminæ of the mediastinum, and finally those which occur in the interlobular fissures of the lungs.

Plastic exudations, when they result either from an acute or a chronic simple process, (by which we mean to imply a process not implicating the exudation,) become transformed into areolar tissue of various degrees of density, into areolo-serous and fibrous tissues, presenting varied relations in reference to form and distribution.

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which the serous effusion is absorbed, in consequence of the fibrous sheath investing the lung, and the destruction of its extensibility and elasticity by continued pressure;—(2), the tendency exhibited by the costal lamina of the fibrous exudation to increase in density and to contract; and (3), the similar tendency on the part of the fibro-areolar tissue that has taken the place of the atrophied muscular tissue in the intercostal spaces.

Contraction, even when arising from general pleurisy, may affect only one portion of the chest; the upper portion may be thus modified, while the lower remains either absolutely dilated, or, at all events relatively so, as contrasted with the sunken portion. Thus, after partial resorption of the effused fluid, an adhesion of the lamellæ of the fibrous exudation may take place, while inferiorly they are separated from one another by the effusion stagnating between them. Partial contractions of the thorax are most commonly the result of partial and circumscribed pleurisies, as we see from the depression of the thorax in the neighbourhood of the clavicles, in consequence of pleurisy being associated with pulmonary tuberculosis around the apices of the lungs; and from the contraction of the lower part of the thorax, in consequence of pleurisies about their base.

The fibrous exudation, and especially its parietal lamella, sometimes undergoes ossification; this change occasionally occurs before, but more generally after, the complete absorption of the serous effusion. The deposition of osseous matter, for the most part, occurs in the thickest portions of the exudation in the form of compact nodulated strings and plates. There are rare cases in which thin osseous plates are formed over the whole pseudo-membranous pleural cone, excepting the thin layer investing the lung, and if this change should occur before the complete absorption of the effusion, this will remain permanently enclosed in an osseous sac.

As some of the above-named causes of thoracic contraction are equally present in pleurisies accompanied by other forms of exudation, so we find these contractions in and after their cure, although, generally speaking, they are not so distinctly marked. Thus, we observe a slight contraction when the visceral lamina of the exudation has become organised into extensible and yielding areolar tissue, and when, notwithstanding that the lung has

regained its original size, the costal lamina has been converted into a thick fibrous sheath; in this case the contraction is consequent on the condensation and shrinking of this lamella. Moreover, after pleurisy with an inconsiderable amount of plastic coagula, and with purulent exudation, we observe thoracic contraction; this is then produced, on the one hand, by paralysis and atrophy of the lung, caused by the long-continued pressure of a large quantity of exudation, and on the other, by the paralysis and consequent histological alteration of the intercostal muscles, which keep equal pace with the intensity and duration of the inflammatory process, and with the quantity and coagulation of the exudation.

Pleurisy sometimes occurs simultaneously on both sides; in these cases both sides may be attacked at the same time, or the pleurisy on one side may succeed that of the other.

Pleurisies with long-standing effusion may give rise to cachexia, general dropsy, hydrothorax on the opposite side, hyperæmia and œdema of the lung of the same side, asphyxia, dilatation of the right side of the heart, venosity, more or less obliteration of the lung, and thus not unfrequently to the eradication of a pre-existing tuberculosis.

There is a *typhous* pleurisy, which, in the strict limitation of the term, is almost always associated with typhous pneumonia.

We have already mentioned, that one of the terminations of empyema consists in suppuration of the pleura; but *this*, and a *similar destruction*, may sometimes arise from the pulmonary parenchyma itself, as well as from without it; in the latter case, abscesses lying on the surface of the thorax, accumulations of tuberculous pus in the sternum and its vicinity, or in the vertebral column, softening encysted exudations on the peritoneum diaphragmaticum, perforating abscess of the liver and mammary gland, ichorous cancer, &c., may be regarded as exciting causes. Perforation of the costal pleura occurs for the most part where there is a tolerably thick and resistant layer of exudation deposited by a previous pleurisy, and adhesion thus established with the lung; there is then no discharge of pus into the thoracic cavity, but it not unfrequently happens that it finds its way through fistulous passages into the adherent lung, and leads to its ulcerous destruction.

1. *Gangrene of the Pleura* occurs in consequence of its being denuded by accumulations of pus or ichor in the costal or pulmonary wall. The pleura then assumes the appearance of a yellowish-white, or more frequently of a blackish or greenish-brown, lax or deliquescent slough, with superficial gangrene of the lung. There is no difficulty in distinguishing it from the acute black softening to which the pleura diaphragmatica is subject from the stomach, or the left layer of the mediastinum from the œsophagus.

c. *Adventitious Products*.—Passing over the areolar and areolo-serous adventitious products, we must notice *anomalous fibrous* and *cartilaginous tissues*, and *anomalous osseous substance*. These are especially frequent in the pleura, and in relation to their origin and their seat they present two distinct varieties. *In one case* they are products of inflammation, which, as has been already stated, become converted into fibroid and cartilaginous tissues, and then into concretions of bone-earth in the form of strings and plates; these are seated on the inside of the pleura, with which they intimately coalesce. *In the other case*, the fibroid and cartilaginous tissues are developed independently of inflammation and merely in consequence of a hyperæmic condition in the subserous areolar and fibrous tissues, and in the tissue of the serous membrane itself. We first observe a whitish, more or less circumscribed opacity and condensation of the serous tissue; there is here a development of tissue resulting in the formation of a smooth or nodular elastic plate, or of a group of granulations of fibrous or fibro-cartilaginous tissue, or even of irregularly-shaped masses, which vary from the size of a pea to that of a nut, and finally ossify. These are always situated under or on the outer side of the serous membrane, and are invested by it.

These two varieties may be easily distinguished from one another: fibrous exudations invest the costal as well as the pulmonary pleura; they ossify, however, only on the costal pleura. Subserous adventitious products occur almost exclusively on the costal and diaphragmatic pleura, and their most common seat is in the intercostal spaces. They sometimes become liberated, and are found free in the cavity of the thorax, in the form of round, nodular masses of cartilage.

2. *Tuberculosis of the Pleura*.—Pleural tubercle occurs in

all the forms in which we have stated that tuberculosis attacks serous membranes; that is to say—

(a), as a rapid metamorphosis (either complete or partial) of a pleuritic coagulum; this is most common on the parietal lamina;—

(b), as tuberculous formation in a pseudo-membranous coagulum that is either becoming organised in some manner or other, or that has become organised to a certain stage; as, for instance, tubercle on and in layers of areolar, fibro-areolar, and other exudations;—

(c), as primary acute tuberculosis in the form of the most minute miliary tubercles.

The second kind of tuberculous formation is very frequently associated with secondary inflammation of the pseudo-membrane serving as the base to the tubercle, and in these cases we may frequently observe hæmorrhagic exudation.

Pleural tubercle is, in most cases, the result of a dyscrasia that has already revealed itself in tuberculosis of a parenchymatous tissue, most probably of the lungs or bronchial glands; sometimes, however, it occurs as the first in the series of successively developed tuberculoses, and not unfrequently it is associated with quiescent or even retrograde pulmonary tuberculosis, indicating a tumultuous recrudescence of the general disease, and from that period being frequently combined with general tuberculosis.

Pleural tubercle not unfrequently softens and gives rise to tuberculous abscesses in the different pseudo-membranous structures in which it is deposited. These abscesses may penetrate the pleura, and even the thoracic walls, with or without caries.

3. *Cancer of the Pleura.*—Cancer of the pleura is much rarer than tubercle. It never occurs as the first in the series of cancerous deposits occurring in an individual, but is always the result of a cancerous dyscrasia that has previously developed itself in other structures. It is very frequently associated with cancer of the mammary gland, with cancer in the mediastina, with cancer of the osseous system, or with general cancerous disease; it is commonly developed simultaneously with cancer of the lungs, and usually runs a very rapid course after the extirpation of large masses of cancer.

The pleura may be perforated from without by adjacent cancerous formations which, after having established the cancerous metamorphosis on it, sprout forth on its cavity in the form of nodular growths; or the cancer may appear primarily on its inner smooth surface, in the form of flattened, roundish or nodular, lardaceous or medullary masses, varying from the size of a hemp-seed to that of a hen's egg, or even of a man's fist. They are sometimes so fluid as to be diffused and form a stratum, and may be either loosely or firmly attached to the serous membrane on which they are situated. In the latter case they involve the tissues of the pleura itself, and in general turn out to be *medullary cancer*.

Their presence always occasions an accumulation of more or less serous fluid in the pleural sacs.

4. *Morbid Contents of the Pleural Sacs.*—In addition to the anomalous contents of the pleural sacs, which have been already mentioned, and to which we shall have occasion subsequently to allude, we shall only at present treat of the presence of gas and serum in the pleural sacs (*pneumothorax* and *hydrothorax*).

There are many sources from which gases of various kinds may accumulate in the cavity of the thorax, and (independently of the atmospheric air finding its way there from without, through a penetrating wound in the walls of the chest, or through the bronchi in injury of the lung with or without an opening in the thorax, or through both causes combined,) a *pneumothorax* may occur under the following conditions:

(a.) In consequence of the opening of a tuberculous abscess before there is perfect adhesion of the lung to the walls of the chest through pleuritic exudation. Here we must particularly direct attention to the abscesses which are preceded by superficial tuberculous infiltration arising from softening.

(b.) In consequence of the softening and separation of a superficial gangrenous eschar of the lungs.

(c.) In consequence of the softening of the so-called metastatic deposits which penetrate the pulmonary pleura before reactive hepatisation can occur.

(d.) In consequence of the opening of a pulmonary abscess communicating with some of the bronchial tubes.

(e.) From the development of gas from the decomposition of purulent and ichorous exudations (*empyema*).

(*f.*) In rare cases, a certain quantity of air is found in the pleural sac, as a product of the inflammatory process, and associated with a benign pleuritic effusion.

(*g.*) In cases in which there is an opening formed into one of the bronchial trunks, in consequence of its corrosion by purulent and ichorous exudation.

(*h.*) In consequence of the laceration of one or more of the superficial pulmonary vesicles in vesicular emphysema, or from rupture of the pleura in interlobular and sub-pleural emphysema.

(*i.*) From perforation of the diaphragm or of the mediastinum, arising from acute softening of the stomach or œsophagus.

When the disease giving rise to the pneumothorax is not in itself fatal, which, however, is the case in softening of the stomach and œsophagus, then the gas accumulated in the thorax invariably causes inflammation of the pleura, and exudation, compression of the lungs, dilatation of the thorax, &c.; the nature of the exudation varying according to the source, nature, and products of the gas, and of the other substances which are simultaneously extravasated into the pleural sac.

Hydrothorax is a very rare disease, if we except those cases in which it constitutes a part of general dropsy; and even then, if we exclude those in which it is dependent on cardiac or pulmonary diseases, it is very rarely the first in order of the various serous effusions. It may arise as a consequence of hyperæmia of the pleura, and in that case the swelling and hypertrophy of the serous membrane are proportional to the duration of the effusion. It is also often present when there are cancerous vegetations on the pleura; but its most common exciting causes are diseases of the heart and large vessels, pericarditis, catarrh and bronchial dilatation, indurated hepatisation, and pleurisy; finally it forms a portion of the general dropsy consequent on diseases of an exhaustive nature, such as typhus, puerperal fever, emaciation from tubercles or cancer, and Bright's disease of the kidney.

When there is a large quantity of air in the pleura, the lung becomes displaced and compressed in the same manner as in pleuritic effusions.

It should be most carefully distinguished from the serous effusion which occurs as one of the processes of inflammation

of the pleura. The inexperienced observer may find some difficulty in the establishment of a correct diagnosis between it and those pleurisies which deposit exudations deficient in plastic matter (the so-called active dropsies), especially when these latter have existed for a considerable period.

III. ABNORMAL CONDITIONS OF THE LUNGS.

§ 1. *Deficiency and Excess of Formation.*

In very imperfect monsters, as, for instance, in cases of acephalia, the lungs, as well as the central organs of circulation, are altogether absent. In lower degrees of monstrosity, and even in cases in which, in other respects, the organisation is normal, there may occur a considerable deficiency of one or both lungs, that is to say, an arrest of development. The development may be arrested at so early a stage, that we can barely observe them as roundish little bodies seated on the bronchi. It generally happens that this condition is a consequence of the contraction of the volume of the thorax, or of pressure exerted on the lungs either by the displacement of the abdominal viscera into the thorax, in cases of absence of the diaphragm, or by the accumulation of fluid in the thorax.

An excessive formation occurs in double monsters, as more or less duplication of the lungs, either with or without a simultaneous duplication of the pleural sacs. The occurrence of an extraordinary number of lobes is apparently due to an excess of development.

§ 2. *Anomalies of Size.—Hypertrophy and Atrophy.*

The manifest differences in the size of the lungs are, for the most part, dependent on the number and the capacity of the air-cells. As an individual structure, the lung will attain a large size when there is a large number of air-cells, and at the same time their volume is well developed; while, under opposite circumstances, the lung will appear small.

The former usually occurs in the male sex, and is generally associated with well-developed muscular and osseous systems, and with a low state of development of the abdominal viscera, whilst the latter is most common in the female sex, and is associated with a weak state of the muscular fibre, fragility of

the bones, and a preponderating development of the abdominal viscera.

The lungs may appear large either within the normal limits or beyond them, when a certain number of the air-cells are dilated; and under opposite conditions they may appear small or abnormally diminished; indeed, the former may occur when the number of dilated cells is comparatively small, and the latter when the number is comparatively great. In the former condition the pulmonary tissue is rarefied, in the latter it is compressed and dense.

Hence in forming an opinion of the size of the pulmonary organs in any individual case, the density of the parenchyma should be especially noted, since it alone determines the respiratory capacity of the lungs; and the two extremes, excessive rarefaction and excessive condensation, constitute very important morbid conditions, of which we shall speak in our observations on changes of tissue.

Hypertrophy of the lungs undoubtedly arises from a remarkable combination of dilatation of the air-cells, with a simultaneous augmentation of their tissues; we sometimes observe it as a vicarious development of one lung, when the other, for any reason, is no longer able to discharge its proper function. It is incontestible that this hypertrophy does not consist in an increase in the number of the air-cells, but in their dilatation, in the increased thickness of their walls, in the enlarged calibre of the capillaries, and in the development of new vessels. The pulmonary tissue is thus rendered denser and more resistant, and the lung exhibits a singular power of resisting external atmospheric pressure; it has become *larger*, and its thoracic cavity wider.

Atrophy of the lungs is the exact reverse of this condition. It is most marked in advanced age, when it is known as *atrophia senilis*; sometimes, however, it occurs in the earlier periods of life, constituting a premature involution of the respiratory organs, and it is only in these latter cases that it strictly falls within the domain of pathological inquiry. It consists in a dilatation of the air-cells (emphysema), and an alteration from their angular or polygonal into a round or elliptic form. The dilatation is the consequence of the emaciation and attenuation of their walls, the vessels of which become

obliterated. The atrophy of the cell-walls may proceed to such an extent that several cells may coalesce; the interlobular areolar structure has then disappeared, and hence the lobular structure no longer exists; the pulmonary tissue then represents an irregular, perforated net-work; the lung itself is of a pale, light grey colour, generally dotted with numerous specks of black matter, conveys a soft downy sensation to the hand, is light and small, and collapses on the opening of the thorax; on cutting into it the air escapes tardily, with a dull and diffused sound. Its tissue is dry and bloodless.

Thus marasmus of the pulmonary organs is, for the most part, associated with an equally well-marked atrophy and dilatation of the trachea, emaciation of its walls and deficient moisture of its mucous membrane, the nature of the changes being in both organs the same. Moreover, as a general rule, it is most highly developed in the superficial portion of the upper lobes, giving rise to a singular displacement of the interlobular fissure, and causing it gradually to assume a vertical position, (Hourmann.)

The thorax becomes depressed over the atrophied lungs, exhibits distinct lateral flattening, and assumes a conical form; there is an arched curvature of the vertebral column in a backward direction; the sternum is thrust forward, and there is a diminution in the vertical diameter in consequence of the curvature of the spine, and the absorption of the intervertebral cartilages, and partially even of the vertebræ themselves. The soft parts about the thorax disappear; the muscles become emaciated and pale; the diaphragm thin, lax, and plicated; and the heart small.

On these changes are based the difficult respiration, and, in a great measure, the collapse, pallor, and lividity of the tissues,—in short, the general atrophy of old age. The impaired state of the respiratory muscles renders the act of inspiration difficult and imperfect; the deficient contractility of the pulmonary tissue, together with the above-named muscular weakness, opposes similar obstacles to the act of expiration; while the surface of the lungs presents to the atmospheric air so obliterated a capillary net-work, that only a small quantity of blood can be submitted to the vivifying process of arterialization.

If this change occur at an earlier period of life, and an

abnormal relation be then established between the degree of involution of the lungs and that of the other organs concerned in hæmatisation, the disease will assume much more importance if active dilatation of the right side of the heart should supervene.

A remarkable enlargement of the lungs takes place in emphysema; a less marked and usually only partial enlargement also occurs in hepatisation, in highly advanced tuberculosis, in cancer of the lungs, &c.

Diminution of the lungs is chiefly induced by contraction of the thorax, the pressure of gases or liquids that have accumulated in the thoracic cavity (pneumothorax, hydrothorax, empyema, &c.), and by obliteration of the bronchi.

§ 3. *Anomalies in Form and Position.*

Congenital anomalies in the *form* of the lungs are, for the most part, confined to some irregularity in regard to the lobes, which present a position of lateral inversion.

Amongst the acquired deviations, we must notice the displacement of the interlobular fissure in atrophica senilis, the pit-like depression of the surface in obsolescence of a portion of a lung, or in cases of cicatrization after loss of tissue, the flattening of a whole lung, or of a circumscribed portion, and the impression produced by the accumulation of gas in the thorax, by circumscribed exudations, aneurisms, or adventitious products.

Again, when the lung has been for a long time surrounded by a pleuritic effusion, and has acquired a thin, although resistant, fibro-serous investment which hinders its perfect re-expansion, it sometimes undergoes a singular change of form. It loses its sharp borders and the concavity of its base, and reminds us of the corresponding alteration of form presented by the liver after inflammation of its peritoneal coat. It sometimes, but not very frequently, happens that, if the pseudo-membranous investment be thinner at some spots than at others, the pulmonary parenchyma bulges out at those places into teat-like processes connected by a pedicle to the base.

Deviations in position, when *congenital*, are seen in the protrusion of the lungs through a wide fissure in the thorax, and in their lateral displacement. Under the *acquired deviations* we must notice the protrusion of the lungs in penetrating

wounds of the chest, their displacement in various directions from dilatation of the abdomen and enlargement of its viscera, from copious effusion in the pericardium, from enlarged heart, from aneurism of the aorta, or adventitious structures in the mediastina, and generally from any kind of accumulation within the thorax. If there be no pre-existing adhesions in the last-named case to oppose the displacement, the lung, as has been already remarked, is constantly pressed inwards and upwards towards the mediastinum and vertebral column. A similar change in position occurs when obsolescence takes place from internal conditions, since the lung is then retracted on the bronchus.

§ 4. Diseases of Texture.

We shall commence with a description of two very simple alterations of tissue, which, although not very striking in themselves, lead to very important consequences, which, singularly enough, resembled each other; these are *rarefaction* (*vesicular emphysema*) and *condensation of the pulmonary tissue*.

a. *Rarefaction of the Pulmonary Tissue.*—*Emphysema.*—Under the term *pulmonary emphysema* we comprehend, according to Laennec, two different conditions, of which one (and by far the more important one) is not fairly entitled to this name; but this inaccuracy leads to no error, because, in using the terms *emphysema vesiculare* and *emphysema interlobulare*, we indicate the seats of the two diseases, and thus distinguish one from the other.

In *emphysema vesiculare* we have a morbid condition of the peripheral portion of the respiratory organs analogous to that which we have already described as dilatation of the bronchi, and even of the trachea. Had Laennec done nothing else for medical science, his discovery of this diseased condition, and of the causes giving rise to it, would have sufficed to render his name immortal.

Vesicular emphysema consists in a permanent dilatation of the pulmonary vesicles, and the respired atmospheric air is actually contained within them, and does not, as in ordinary emphysema, become extravasated into the interstitial texture.

It not unfrequently arises very rapidly as a vicarious development of pulmonary parenchyma in cases where a great portion of the lung has become impermeable; and it appears

more especially to be produced in a high degree during the last moments of existence, as a consequence of the laboured inspirations which then occur. Thus in diffused hepatisation we find the edges of the inflamed lobes puffy and emphysematous, and in the higher degrees of pulmonary tuberculosis, the same condition is observed in the interstitial parenchyma between the tubercles, and in the superficial stratum of the lung. In like manner it develops itself as a sequence of those acute and chronic diseases which prove fatal by paralysing the nervous apparatus presiding over the chemical process, and in which there is the most laborious action of the respiratory muscles, deep inspiration, and an insatiable thirst for air; or it arises as a consequence of a sudden check to expiration, as in hæmorrhages from the air-tubes, when the bronchi become obstructed by blood.

In such cases we observe the following appearances: the emphysematous portion of the lung is puffed up, and conveys to the hand a peculiar feeling, which may be compared to that of a cushion filled with air; is pale, varying in tint from a palish red to a dull white colour, and is perfectly anæmic; is dry, collapses rapidly on being cut, but on pressure crepitation is indistinct and dull; it floats on the surface when placed in water; its cells are more or less dilated, and their walls are attenuated in proportion to the rapidity with which the morbid change has been developed. Finally there is sometimes extensive laceration of the dilated cells, and the emphysematous portion of the lung then presents the appearance of a torn network swollen with air. This form of emphysema seldom attains this degree, except on the anterior edge and towards the base; and at these parts it gives rise to the escape of air beneath the pulmonary pleura, which consequently peels off from the lung.

This form of emphysema, in so far as relates to the attenuation of the walls of the dilated cells, presents an analogy with senile atrophy of the lungs.

Another form of vesicular emphysema develops itself slowly, gradually spreading itself over a large portion of the lung, till it finally involves the whole organ; it arises, in part, from other causes than those already mentioned, and constitutes a substantive disease of the lung, which, as Laennec remarks, unquestionably gives rise to most of the so-called nervous asthmas.

It presents many varieties in degree and extent. By degree, we refer to the extent of the dilatation of the pulmonary cells; it must, however, be remarked, that in emphysema of long standing, we always simultaneously find several degrees of dilatation, and that it is only during the commencement of the disease that the dilatation is observed to be uniform. The pulmonary cells may be dilated to the size of a millet-seed or pin's head, or to that of a hemp-seed, a pea, or even a bean, and, in proportion to the size which they attain, they deviate the more from their original shape. At first the disease is a genuine, simple dilatation of the cells, and when the cell-walls become to a certain extent thickened and rigid, it may be regarded as an active dilatation of the cells somewhat analogous to hypertrophy of the lungs. In higher degrees, on the contrary, the dilated cells unite to form larger spaces, their walls becoming atrophied by the pressure they exert on one another. Such hemp-seed, pea, or bean-sized cells always present a very irregularly sinuous, but on the whole a roundish form, and exhibit a singular internal arrangement; for on their inner surface there are elevated ridges, projecting to various heights, within the cavities of the dilated cells, traversing them in various directions, and forming boundaries and imperfect partition-walls to the different sinuosities. We likewise perceive delicate threads, either extending across the cells, or hanging free in their cavities: these cover the elevated ridges and the remains of the contiguous walls of the pulmonary cells. The pressure exerted on the adjacent tissues, which gives rise to their atrophy, is proportional to the dilatation of the cells; and the cell-walls becoming thick and rigid, the emphysematous lung, when a section is made, either does not collapse at all, or collapses very slowly.

Moreover, this form of emphysema occurs most frequently, and is most highly developed, in the peripheral portion, and along the edges of the lungs: it is not unfrequently associated with bronchial dilatation; and this, amongst other signs, establishes the affinity of these two diseases. It either attacks a small portion of the lung only, being confined especially to the anterior edge of one or other of the upper lobes, or else it spreads over a whole lobe, or a whole lung, or even both lungs.

In cases of emphysema of both lungs, the association of

all the anatomical signs presents the following picture of the disease :

Barrel-shaped dilatation of the thorax, with permanent depression of the intercostal spaces ; great dorsal curvature of the spine ; hypertrophy of the respiratory muscles ; and a clear sound on percussion.

On opening the thorax, the lungs expand beyond the walls of the chest, are seen to be remarkably large, and do not collapse under the pressure of the atmosphere.

On their surface, and especially at their anterior edges, we find round prominences as large as a hemp-seed or a pea, either standing alone or arranged in groups, and which are nothing more than the dilated pulmonary cells which have been already described.

The lungs have a very peculiar, soft, elastic feeling, which may be compared to that of a cushion filled with down.

On being cut they collapse very slowly, and the air escapes sluggishly, with a very diffused sound, scarcely amounting to crepitation, and somewhat resembling that of air slowly escaping from a pair of bellows.

Their tissue is pale throughout, anæmic, and singularly dry.

When only one lung is emphysematous, then only the corresponding half of the thorax is dilated ; but an important fact in this case is the displacement of the mediastinum and the heart towards the opposite side. Finally, if only individual portions of the lung are emphysematous, they may, if they are very numerous, and the disease is highly developed, prevent by their pressure the expansion of the neighbouring healthy cells, and thus retain them in a state of persistent compression.

The conditions giving rise to the production of emphysema, and its pathogeny in general, although much labour has been devoted to the affection, are still far from clear. Laennec regards it as a consequence of his so-called dry catarrh with pearl-coloured secretion, and explains it in a mechanical manner : this secretion, and the catarrhal puffiness of the mucous membrane, obstruct the bronchi in such a manner, that although they allow the inspired air to enter the pulmonary vesicles, the diminished energy of the act of expiration presents an impediment to its escape, and hence a portion of it is retained. In the succeeding inspirations new air is again

conveyed to the pulmonary vesicles, whose dilatation is thus effectually accomplished, the expansion of the inspired air, from its elevated temperature, doubtless contributing to the result. Moreover, a prolonged retention of the breath during parturition, at stool, or in blowing wind-instruments, may give rise to emphysema.

In opposition to the view that emphysema is a consequence of catarrh, cases have been adduced in which either no catarrh had existed, or when it only followed the dyspnœa as a symptom of pre-existing emphysema; in reference to the last-named causes, it is alleged to have occurred in persons who have never been exposed to these diseases. Hence, a spontaneous dilatation of the pulmonary vesicles has been assumed, which at one time occurs as premature atrophy with attenuation of the cell-walls, while at another time, from equally unknown causes, it is associated, as in other hollow organs, with hypertrophy of the cell-walls.

Laennec's view regarding the former mode of development of the affection, is essentially an important one. We do not, however, believe that the long retention of the breath is, in itself, the principal cause of the forcible expansion of the air-cells; we are much more inclined to attribute it to the very deep and forcible inspirations which at length follow the expirations, and, in illustration of this view, we may refer to the nature of the inspirations in croup, in the bronchial catarrh of children, and in whooping-cough. Besides inducing forcible dilatation, they may also cause a paralysis of the contractility of the pulmonary tissue, and, consequently, a stagnation of air in the dilated pulmonary vesicles.

Emphysema may, however, be developed in cases where none of these injurious influences have been present; and it may occur gradually in persons leading a sedentary life. In these cases the less frequent but the proportionally deeper inspirations are the more to be regarded, because they take place with very little action of the diaphragm (abdominal respiration), as the occupation of such persons requires a bent position compressing the abdominal cavity, and, at the same time, powerful exercise for the arms. A paralysed and atrophied condition of the diaphragm is here of the greatest importance; for the hinderance to the abdominal respiration thus induced is compensated by

the strained activity of the other great respiratory muscles; and this circumstance is in accordance with the facts that the dilatation of the thorax is most marked in its upper segment, and that emphysema is primarily and most fully developed in the upper lobes, and especially in their anterior portion.

The thickening of the walls of the dilated air-cells arises, in our opinion, principally from the tissue adjacent to them becoming broken down by compression, and fusing with the cell-walls themselves; nevertheless if the dilatations increase from the persistent pressure exercised by the dilated cells on one another, atrophy of the contiguous walls will ensue, and the cells will unite to form larger cavities, much in the same way as we occasionally observe to occur in contiguous bronchial sacs.

The dyspnœa, conditional on emphysema, depends on several causes.

a. The excessive accumulation of air in the pulmonary vesicles hinders the proper filling of the capillaries ramifying on their walls by the pressure it exerts on them, and thus interferes with the vitalisation of a sufficient quantity of blood.

β. In the higher degrees of emphysema numerous capillaries become obliterated, not only in the walls of the dilated cells, but also in the surrounding atrophied tissue,—a condition which induces the above consequence in a still higher degree.

γ. The diminished contractility of the pulmonary tissue and the constantly laboured inspirations which then become necessary in consequence of the imperfection in the chemical process of respiration, allow of only a very imperfect emptying of the pulmonary cells, and consequently give rise to the permanent stagnation of air no longer fit for the purpose of respiration, which, in its turn, also tends to prevent a sufficient vitalisation of the mass of the blood.

The impermeability of the capillaries depending on the two first-named conditions gradually, but unfailingly, leads to disease of the right side of the heart in the form of active dilatation, which proceeds to affect the venous system; the venosity and cyanosis which ensue from these changes constitute the leading grounds for the immunity of asthmatic persons from tuberculosis.

The impermeability of the capillary vascular system, moreover, gives rise to the anæmic state of emphysematous lungs,

thus rendering it an impossibility that œdema, stasis, hæmorrhage, or pneumonia, should be developed in them.

It is easy to understand how it proves fatal. It kills by finally inducing paralysis of the lungs, by asphyxia from the accumulation of air no longer fit for the process of respiration, by paralysis of the heart, or by vascular apoplexy of the brain.

Emphysema interlobulare is the only form which, strictly speaking, deserves the name of emphysema; it consists in an accumulation of air in the cellular interstices of the pulmonary lobules. It can only result from the rupture of one or more pulmonary vesicles, and the escape of air from them into the adjacent cellular interstices, if, indeed, we except the spontaneous development of gas in the interlobular cellular tissue, which is not altogether impossible.

We consequently find air-bladders in the cellular interstices, and especially on the surface; they vary in number and size, and are characterised by their paleness, transparency, and round or rather oblong form; they may be made to move in the direction of the interstices, and to run into one another, so as to form ridges which ramify in the same direction superficially, and into the body of the lung; sometimes they circumscribe and, as it were, insulate the lobules, and as they are broadest on the surface, and as their size diminishes in proportion to the depth to which they penetrate within the substance of the lung, they present a wedge-like shape. When they are very small and closely crowded together, they present the appearance of froth. On making a section of a portion of dried lung, we find the interstitial tissue presenting irregular cellular spaces of larger or small size, heaped, without order, on and around one another, and perfectly different from the adjacent air-cells. As has been already mentioned, most of the air is usually found accumulated in the peripheral interstices, so that the pleura presents a puffed up, vesicular appearance. The air often makes its way into the cellular tissue uniting the pleura to the lung, peeling off large patches, and forming flattish, convex, moveable air-bladders; and, in these cases, it is to be feared that some of these bullæ may be ruptured, and that the air may be extravasated into the pleural sac. In other cases the extravasated air may penetrate into the substance and towards the root of the lungs, and pass into the cellular tissue of the

mediastinum, and from thence into the neck, and thus cause general emphysema.

This condition usually co-exists with a puffy state of the lungs, but never with well-marked vesicular emphysema. It is most common in children; and is occasioned in them, as well as in the rarer instances in which it occurs in adults, by very rapid, deep inspirations, or by long retention of the breath when great muscular exertions are made, requiring a fixed condition of the thorax. It is most commonly situated in the upper lobes, and especially along their anterior edges.

b. Condensation of the Pulmonary Tissue.—A certain degree of condensation is natural to the lungs of children; it sometimes occurs in adults as an individual peculiarity, and is then often associated with smallness of the lungs and pleural sacs. It is also present as a transitory condition during pregnancy.

It only comes within the limits of pathology, when it has become permanent and highly developed, and offers a persistent impediment to the capillary circulation through the lungs.

Such a degree of condensation may arise when the abdomen becomes enlarged and encroaches on the thoracic cavity, but in children it is more frequently dependent on lateral depression of the thorax consequent on atrophy of the great respiratory muscles, or on rachitis affecting the chest; it may also arise from spinal curvature, distension of the pericardium, enlargement of the heart, large aneurisms, adventitious products, &c.; or from the pressure exerted by an accumulation of air or fluid in the cavity of the chest, from pleuritic exudation, or from bronchial dilatation; and, according to the various exciting causes, it may occur simultaneously in both lungs, or only in one, or merely in certain portions of the pulmonary tissue, as in cases of rapidly developed emphysema, where we not unfrequently find single lobules compressed in the centre of the emphysematous portion, or in cases of atrophy of the external respiratory muscles, where single circumscribed portions of lung are found in a state of condensation under the bent anterior ends of the ribs.

There are different grades varying from simple increase of density characterised by augmented consistence and compression of the pulmonary tissue, and by a stasis and hyperæmia depending on obstructed circulation, to such a degree of compression as to

destroy the air-cells, to arrest the capillary circulation, and to give rise to atrophy of the texture of the lung.

The most intense compression of the lung occurs in cases where there is abundant pleuritic effusion. With the alterations in position and form, to which allusion has already been made, the lung always becomes denser, and gradually becomes impermeable to air, and, finally, even to the passage of blood along its capillaries. If the lung still contains blood, its red colour gives it such a similarity to flesh, that this condition has received the name of *carnificatio pulmonis*, but at a subsequent period it becomes of a dirty brown, or, more commonly, of a bluish-grey or lead colour, and is tough and leathery, and sinks in water.

If the state of extreme compression persist for a length of time, the pulmonary tissue finally becomes obsolete, that is to say, it becomes converted into a cellulo-fibrous tissue,—a condition altogether distinct from atrophy of the pulmonary tissue.

Excessive condensation of the lungs gives rise to consequences similar to those of emphysema; it impedes the capillary circulation, and thus occasions stasis in the trunk of the pulmonary artery, giving rise to active dilatation of the right side of the heart, and consequently to venosity and cyanosis. Hence, like emphysema, it affords a remarkable immunity from tuberculosis, especially when associated with curvature of the spine.

There is a peculiar form of anomalous condensation of the pulmonary tissue, probably dependent on a congenital bronchial catarrh or catarrhal pneumonia, and consisting in a deficient development of the lungs of new-born children, in which certain portions of those organs retain their foetal condition after birth. It is termed *atelectasis* of the lungs, and presents various degrees of obstruction to the closure of the foetal passages, namely, the ductus arteriosus and the foramen ovale, thus giving rise to predominance of the right side of the heart and to cyanosis.

c. Hyperæmia ; Stasis—Apoplexy of the Lungs.—No organ, with the exception of the brain, is so frequently the seat of hyperæmia as the lung. It occurs in various degrees, and develops itself either gradually or with intense rapidity, and is the anatomical basis of most sudden deaths.

In a lesser degree, as simple hyperæmia, it is frequently an habitual, and not rarely a periodic affection of an active nature; it often ensues with great rapidity, and may prove speedily fatal by itself, or more frequently by the superaddition of acute œdema. We then find both lungs uniformly puffy, and of a dark red colour; their vessels, even to the capillaries, being filled with dark blood, and their tissue being succulent and softened, but still crepitating. In the bronchi we find a greyish, sometimes reddish mucus mixed with air-bubbles. The heart is usually somewhat dilated, and always contains a large quantity of thin liquid or slightly coagulated dark blood, especially in its right cavities. The veins of the membranes of the brain are usually full to distension, and serous effusion into the cerebral ventricles frequently occurs as a consecutive complication. The outer surface of the body is characterised by livor and by the rapid occurrence of extensive and very dark-coloured death-spots; the face in particular is very puffy, and of a more or less bluish tint; the eyes and mouth are generally more or less open, and the conjunctivæ injected; the mucous membrane of the mouth is livid, and that of the throat is covered with tough mucus. Greyish or pale reddish, frothy mucus is found in the trachea.

In a higher degree hyperæmia amounts to *stasis*. In this stage the parenchyma of the lung is of a purple or black-red tint, and, as it were, saturated with blood; and as it somewhat resembles the substance of the spleen, this condition has received the name of *splenization*. Several of the other characters of this condition are subject to various modifications, depending primarily on the degree and the duration of the hyperæmia, but to a lesser extent on the nature of the stasis and the composition of the blood. When it is recent and comparatively slight, the parenchyma is denser but easily torn; it crepitates, although less clearly than in the normal state; on cutting it, a large quantity of fluid blood escapes; the diseased portion is puffy, and floats on water. In a higher degree, and when the stasis has continued for a longer period, the walls of the air-cells and the interstitial tissue become swollen, so that the former become perfectly impermeable to air; the parenchyma consequently become denser, hard, and heavy, and ceases to crepitate; and on making an incision

only a comparatively trifling quantity of thick fluid blood escapes. The blood appears, as it were, fused into the tissue of the lung, the whole affected portion having a somewhat shrunken appearance.

The blood contained in the splenified portion of the lung presents various shades of discoloration, viscosity, fluidity, or gaseity, according to the nature of its composition and the character of the stasis.

According to circumstances we occasionally find in the bronchial tubes either a sanguineo-mucous or sanguineo-serous fluid.

Stasis is the result of an active or passive, or of a mechanical hyperæmia; either of these may prove fatal by itself, especially when extensively developed; and either may sooner or later pass into inflammatory stasis and inflammation. It never attacks both lungs simultaneously and in an equal degree; it generally exhibits a preference for the lower lobes, and when it extends over a whole lung, usually commences inferiorly.

Stases of a *passive* nature in the most dependent posterior and inferior portions of the lungs, such as are developed in bedridden old persons, or in individuals confined to bed for a length of time in consequence of cerebral disease, typhus and typhoid affections, any adynamic diseases, and especially paralysed conditions of the lungs, are important. They constitute the *pulmonary hypostasis* of Piorry.

Mechanical stasis is most commonly dependent on organic diseases of the heart, although excessive density of the lungs may give rise to it. It is developed, according to circumstances, either in the arterial or the venous portion of the capillary system of the lungs.

It is of great importance to distinguish, as clearly as possible, between these conditions and the stasis which is developed in the body after death,—cadaveric hyperæmia of the lungs, more especially as this is very frequent and is often combined with the former. The latter is always most marked at the posterior portion of the lungs, gradually diminishing in the superior and anterior directions. The lung is soft and crepitates, and apparently is not so much saturated with actual blood, as with a sanguineous, dark-red, frothy, discoloured serosity, which is poured forth in abundance from the cut surface, and may be entirely removed

by moderate pressure, after which there remains a pale, discoloured parenchyma compressed in proportion to the pressure employed. In consequence of prolonged imbibition, the pleural sacs not only become discoloured, but a certain quantity of sanguineous discoloured serosity makes its way into their cavities.

Different views have been held, especially in recent times, since doubts have been entertained regarding Laennec's theory of hæmoptoic infarctus, respecting the relation which these conditions bear to pulmonary hæmorrhage and apoplexy of the lungs. For our own part, on the one hand, we regard them as representing *hyperæmia* of a lower or higher degree, which, under certain conditions, may give rise to hæmoptysis, and, on the other hand, it appears clear to us that they also represent *apoplexies*, namely, in a less degree a vascular apoplexy, and in a higher degree an apoplexy with effusion of blood into the parenchyma of the lungs; but as the blood is not at all events originally effused into the cavities of the air-cells, apoplexies are *not necessarily* associated with hæmoptysis, and hence must be distinguished from it.

The latter variety, namely, hæmorrhage into the cavities of the air-cells, corresponds with Laennec's *pulmonary apoplexy* or *hæmoptoic infarctus*. Our own experience confirms the views of that great physician regarding the existence of this morbid change, and the manner in which it is produced. When highly developed, it is attended with laceration of the texture of the lungs.

The apoplexy of Laennec is characterised by the following signs:—

We find blackish-red patches in the substance of the lungs, which attract attention not only by their colour and consistence, but also by their definite outline. On examining the cut, or, what is better, the torn surface of the diseased portion, we observe it to be more or less coarsely granular and dry, the granulation being often very irregularly distributed. The tissue itself is tough and yet easily torn, and presents throughout, both at the centre and at the periphery, the same consistence. The whole represents an effusion of blood into the cavities of the air-cells, which distends them to a certain extent and then coagulates within them, thus giving rise to the granular texture of the hæmoptoic infarctus. The interstitial tissues are

compressed, and infiltrated with blood, and hence the colour of the diseased part is uniform throughout. The terminations of the bronchial tubes are also filled with the extravasated blood, their walls being reddened by imbibition, just as we observe in the case of blood-vessels. On scraping the infarctus with the back of a scalpel, there is poured forth a very slight quantity of thick blood intermixed with numerous, black, grumous flocculi.

Hæmoptoic infarctus bears the greatest similarity to red hepatisation of the pulmonary tissue; none but very inexperienced persons can, however, mistake one for the other, for each of the above properties of infarctus presents a distinguishing sign from hepatisation. These, briefly summed up, are the well-defined limitation of the infarctus, the homogeneity of its consistence and colour throughout its whole extent, the *coarse and irregularly granular* appearance and the dry fragility of its cut or torn surface, and the nature of the product obtained on pressing or cutting its surface.

The pulmonary tissue in contact with the infarctus is either in a perfectly healthy condition or else in a state of some other pre-existing or consecutive disease; in every case it is clearly separated from the infarctus. Amongst the pre-existing diseases we must especially mention tuberculosis and pneumonia, whilst the most common consecutive affections are emphysema and œdema of the lungs.

It occasionally happens that this limitation of the hæmoptoic infarctus cannot be detected without a somewhat close examination. This is the case when the parenchyma surrounding it to a certain distance, is the seat of an effusion of fluid blood, whose limitation is by no means sharply defined, since it changes towards its periphery into a palish, sanguineo-serous infiltration, and thus gradually loses itself in the normal tissue. Still, by a careful investigation, we may discover the infarctus seated within the fluid effusion, and plainly separated from it by its consistence and darker colour.

The size of the hæmoptoic engorgement is seldom very great, for while, as Laennec observes, it scarcely ever exceeds four cubic inches, it is frequently less than one. We often find only one infarctus; sometimes, however, several are simultaneously present in one or both lungs.

They are deeply seated in the parenchyma of the lungs, near

their roots, or in the posterior portion of the lower lobes; they are, however, occasionally found near the surface, and may be recognised through the pleura by external inspection. It sometimes happens that when they have existed for a considerable period, the pleura above them becomes inflamed.

They are often, but by no means always, accompanied by considerable hæmoptysis; their size stands in no relation to its amount, indeed there may have been very considerable hæmoptysis, without a trace of hæmoptoic infarctus being perceptible after death; thus, when the effused blood has coagulated rapidly and completely in the pulmonary cells, notwithstanding the hæmoptoic infarctus, there will be no hæmoptysis; in another case the blood does not coagulate at all, but is coughed up in a fluid state, and then, notwithstanding the hæmoptysis, there is no hæmoptoic infarctus; or, again, the primary effusion may coagulate and form an infarctus while hæmorrhage in the surrounding parenchyma may be the source of hæmoptysis, (see above.)

This form of apoplexy is very frequently found to be associated with active dilatation of the right side of the heart, and it seems to bear the same pathogenetic relation to this cardiac affection as cerebral apoplexy bears to active dilatation of the left side of the heart.

In recent times some doubts have been suggested regarding the true connection between hæmoptoic infarctus and pulmonary hæmorrhage, and it has been regarded as the result of hæmorrhage of the finer bronchial ramifications, that is to say, as depending on the coagulation of blood which has escaped from the bronchi into the pulmonary vesicles. Although we fully believe that this may sometimes be the case, yet in the absence of any positive proof we prefer adopting Laennec's view, in relation to the assumed bronchial hæmorrhage, for the following reasons: (1), because hæmoptoic infarctus very often occurs without hæmoptysis, while a bronchial hæmorrhage could hardly take place without any sanguineous expectoration; and (2), because if, as is reasonable, we recognise the influence of hypertrophy of the right side of the heart, we shall see that this influence, notwithstanding the anastomoses of the two systems of vessels, is especially exerted on the pulmonary arteries, and that it will thus serve to elucidate true pulmonary hæmorrhage.

When this form of apoplexy is very much developed it is

accompanied with *laceration of the pulmonary tissue*; we find a cavity in the lung similar to those which are met with in cerebral apoplexy, and containing a certain quantity of more or less coagulated blood. The surrounding pulmonary texture is torn, suffused with blood, and presents, to a certain degree of thickness, an appearance of hæmoptoic infarctus.

The position of these cavities coincides with that of the hæmoptoic infarctus; it has, in rare cases, happened that when situated in the peripheral portion of the lungs, they have opened by a rent into the pleural sac, thus giving rise to the free effusion of blood into that cavity, and to pneumothorax.

The size of these cavities varies, but it scarcely ever exceeds that of the hæmoptoic infarctus. Gangrene of the lungs sometimes, however, gives rise to very considerable accumulation of blood.

Simple hyperæmia and stasis are easily reduced to the normal state, especially under proper and judicious treatment; but they leave a great predisposition to relapses, and hence they usually require a prolonged prophylaxis.

The following questions suggest themselves:—what alterations do hæmoptoic infarctus and apoplexy with laceration undergo in the progress of time? and in what way is the tendency to cure and its successful accomplishment evinced?

It is only very rarely that experience presents us with pure indisputable facts bearing on the various stages of the healing process, necessary for the solution of these questions; still from the scanty materials in our possession, and by a comparison with analogous processes in other organs, we arrive at the following conclusions:—

The effusion in hæmoptoic infarctus either (1) quickly becomes fluid, assumes a blackish-brown, rusty, and wine-lees tint, and in this state is partly absorbed and partly excreted through the bronchi, (thus, doubtless, causing the peculiar expectoration sometimes observed to follow hæmoptysis,) the parenchyma remaining for a time moist, soft, lacerable, and of a rusty or wine-lees colour, and gradually returning to its normal state; or (2) the effusion is only partly removed in this manner, and there remains a tough fibrinous coagulum which gradually becomes perfectly decolorised, or a loose glutinous coagulum saturated with black pigment, the surrounding parenchyma

becoming shrivelled up, and degenerating into a cellulo-fibrous tissue of either a white or a blackish tint.

Apoplexy with laceration heals, after the absorption of the effusion, either by a direct agglutination of the walls of the sac, or by the contraction of the parenchyma round a fibrinous coagulum, which finally become cretified, or by the conversion of the parenchyma into a cellulo-fibrous capsule, enclosing a glutinous coagulum consisting, for the most part, of pigment.

d. Anæmia of the Lungs.—There are various conditions which may give rise to a deficiency of blood in the lungs. It may depend:—

a. On exhausting hæmorrhages.

b. On wasting of the blood, consequent on various acute and chronic diseases.

c. On inspissation of the blood, consequent on rapid and great loss of serum, and on the inability of the blood, in this condition, to enter the capillaries; this is especially the cause of the anæmia of the lungs in asiatic cholera.

d. Finally it occurs in association with pulmonary atrophy, with emphysema, and with the higher degrees of compression of the lungs.

e. Edema of the Lungs.—Pulmonary œdema is a very frequent and extremely important disease. Its essential and primary symptom is the infiltration of the parenchyma with a serous fluid, which is obvious even from an external inspection, and much more so on examining the interior of the viscus, which pours forth a serous fluid when a section is made into it. The serum, however, does not vary only in regard to its quantity (that is to say, not only are there differences in the degree of the œdema), but it likewise presents many differences in relation to its properties.

In order to understand the importance of pulmonary œdema under all conditions, it is necessary for us to direct attention to the information which we have acquired from clinical observation, and from careful examination and experimental investigation of the dead body in relation to the seat of the serous effusion. We thus ascertain that the serum is effused into the cavities of the air-cells, where it accumulates, either alone or mixed with varying quantities of air, according

to circumstances. From hence it flows in greater or less quantity, either mixed with air and frothy (as bronchial foam), or unmixed with air, into the bronchial tubes. The walls of the air-cells and the interstitial tissue are also more or less saturated and infiltrated with serum, but the true seat of the fluid which so often escapes in astonishing quantities from the cut surface of the parenchyma of an œdematous lung, is in the air-cells and the bronchial canals.

Pulmonary œdema occurs both in an *acute* and in a *chronic* form, and between these extremes there are many transition stages presenting mere shades of difference. In *acute œdema* the lung appears swollen, does not collapse, feels puffy, and when we press it with the finger we detect a fluid which escapes with a crackling noise; its elasticity is only slightly diminished, so that scarcely any perceptible pitting remains after the pressure; it is of a pale reddish colour, very pale and deficient in blood when anæmia is present, and more or less red and congested if there be hyperæmia; the serum which is effused from the cut surface is mixed with much air, which renders it frothy, and is usually of a pale red colour; but in œdema arising from prolonged stasis and simultaneous decomposition of the blood, it is red and discoloured, having an icteric tint. The parenchyma is softer than usual, very moist, singularly yielding, and easily torn.

If the œdema lasts *for a longer time*, the pulmonary tissue gradually loses its elasticity, the lung pits more distinctly on pressure, becomes paler, assumes a faded, dirty grey colour, and becomes opaque and dull; the air is gradually pressed out of it; it crepitates less, when cut; and the serum is less frothy, gradually loses its colour, and becomes clear and limpid. The parenchyma becomes gradually infiltrated with serum, the walls of the air-cells and the interstitial tissue become swollen, and hence the lung becomes denser and more resistant.

Finally, in cases where *chronic œdema* has been very fully developed from its commencement, the lung appears pale, of a dirty grey colour, anæmic, not swollen, but heavy, dense, and resistant, pitting on pressure and no longer crepitating; a greyish or somewhat greenish serum unmixed with air flows from the cut surface. Dropsical accumulation in the pleural sac is almost always simultaneously present.

Œdema of the lungs, like acute œdema of the glottis, is often *very rapidly* developed; from an active hyperæmia or a passive or mechanical stasis, it quickly reaches a high degree of intensity, extends simultaneously over both lungs, and in a short time causes death by suffocation. This is frequently the cause of the suffocation of adults and of new-born children, and is often combined with hyperæmia and serous effusion within the cavity of the cranium. The dead body usually presents the same appearances as those which we have described as occurring in pulmonary apoplexy; the lungs in particular exhibit œdema, and a frothy serous fluid is accumulated in the bronchial passages, which is frequently seen as a thick white, or whitish red froth, at the oral and nasal cavities. It may also be developed as a consequence of acute or chronic bronchial catarrh, or of exudative processes (croup) on the tracheal and bronchial mucous membranes; it is a constant symptom in acute pulmonary tuberculosis, in acute decompositions of the blood and after the retrogression of erysipelas, scarlatina, variola, rheumatism, miliaria, &c. In the form of more or less developed acute œdema it accompanies the various stages of pneumonia and the metastases; and is associated with hæmoptoic infarctus, with pulmonary cancer and especially with pulmonary tuberculosis. Lastly, it appears as a consequence of cerebral diseases, of general anæmia and tabes, and occurs towards the end of almost all chronic diseases.

Chronic œdema, moreover, exists with general dropsy, with dropsy of the great serous sacs, with chronic diseases of the heart and great vessels, &c. It is rarely an idiopathic and independent disease.

The extent of œdema is various; the very acute and rapidly fatal œdema generally attacks both lungs almost equally; in other cases it is limited to individual portions of them. The œdema in cases of pneumonia commonly affects the circumference of the inflamed part; that which occurs as a consequence of chronic diseases, for the most part, attacks the posterior and inferior parts of the lungs, which are most exposed to the influence of gravitation.

f. Inflammations of the Lungs (Pneumoniæ).—Pathologists are in the habit of recognising only one form of pneumonia. It is true that this is by far the most frequent form; but even in

regard to this there are several points in which we cannot agree with the accepted view. We may provisionally and very briefly remark that the evidence of its croupous nature will be the more manifest in proportion to the epidemic constitution and the special cause of the disease, the rapidity of its course, the degree of its intensity, &c. We shall treat of this, the most common form of pneumonia, under the designation of:—

1. *Croupous Pneumonia*.—The course of this disease is divided, as is well known, into three stages which have received the names of *inflammatory engorgement*, *hepatisation*, and *purulent infiltration*. We shall first consider the case in which a whole lung, or at least a whole lobe, is affected.

The first stage, inflammatory engorgement, is always preceded by the above-described condition of simple stasis and splenisation of the parenchyma; but, conversely, this condition is not always developed into inflammatory stasis or engorgement. This affords the explanation of the contested question regarding the inflammatory nature of simple stasis and its significance as a stage of the inflammatory process. It is only by a careful examination that we can distinguish inflammatory engorgement from simple stasis. The lung is generally of a dark red colour, heavy and tough; it pits on pressure, and we perceive that it contains a fluid and little or no air. On cutting it, we find its substance denser than in the normal state, in consequence of the swollen condition of its tissues and of its being filled with a sero-sanguineous fluid; and according to the degree of this state the lung may either crepitate and swim in water, in consequence of its still containing a little air, or it may sink and not crepitate; it is easily torn, very moist, and pours forth a sero-sanguineous fluid, which is sometimes rather frothy and sometimes not at all so.

This condition has, as we have already remarked, the greatest similarity and affinity to simple stasis, especially when the latter is combined with œdema.

We will now direct attention to the characteristic symptoms by which inflammatory engorgement may be distinguished from the above-named similar condition. Amongst them we may mention the *colour tending to a brownish red*, and the *moisture* of the parenchyma, which in itself is sufficient to distinguish the inflammatory from the simple stasis, and also from that

combined with œdema, by the special circumstance of its depending on the tissue being filled with blood that has already undergone the inflammatory metamorphosis, or, in other words, with a brownish or brick-red, thin but viscid fluid mixed with black, crumbling flocculi. As soon as the transition to the second stage commences, there is a *secretion* of a very viscid, tough, reddish brown fluid,—the characteristic sputum, as may be proved by an examination after death; and, finally, there is the true exudation with which appears—

The second stage, or that of *hepatisation*, in which the lung appears both externally and internally of a dark brownish red colour, is solid but friable, does not crepitate, and sinks when placed in water. On examining the cut surface we either observe the above colour uniformly distributed, or it is deposited in the form of irregular spots amongst the black pulmonary tissue; while the pale red interlobular tissue presents ramifications, and the whitish bronchial tubes and the blood-vessels form stripes or islands which destroy the uniformity of the colouring, and give the cut section a marbled appearance. Further, the cut or torn surface presents a change of texture which is perfectly characteristic; when the light falls obliquely on this surface we perceive that it has a granular appearance, which is the special reason why it resembles the tissue of the liver, although the similarity is aided by the firmness, fragility, and colour of a hepatised lung. Hence the origin of the term *hepatisation*, which is now generally adopted and understood. The character of the granulation is uniform, and the individual granules are roundish. Scarcely anything exudes from the cut surface, and it is only by a certain amount of pressure, or by passing the scalpel over it, that a brownish red, turbid, sanguineo-serous fluid escapes, mixed with blackish brown and a few reddish grey flocculi.

The volume of the hepatised lung does not in general exceed that of the healthy lung in a state of full inspiration; hence its surface is smooth, and never indented by the ribs, and there is no dilatation of the thorax. Sometimes, however, we find single lobules projecting higher above the surface than others, in consequence of a want of uniformity in the progress and the degree of the exudation; and the granulation of these tissues is coarser in consequence of the products of inflammation being here deposited in greater quantity than in other parts.

This form of hepatisation is named the *red*, to distinguish it from those varieties in which this colour is no longer present, although the granular texture remains.

On what does the granular texture of the hepatised lung depend? This is a most important question; the ordinary answer to which is, that it results from so great a swelling of the walls of the air-cells that their cavities become obliterated, each granulation being thus represented by an air-cell. We can by no means give our assent to this generally received opinion, for we are convinced that the granulations are produced by the inflammatory product deposited in the cavities of the air-cells; we shall, however, postpone bringing forward our evidence on this point, since the perfect solution of the question is intimately connected with the determination of the seat and nature of the pneumonic process.

Each granulation is a hardish, fragile, dark-red, roundish plug, which adheres so closely to the dark-red, swollen wall of the air-cell, that it is difficult to separate and extract it.

Pneumonia passes from the stage of red hepatisation through several scarcely distinct transition-stages till it finally attains the true *third stage*. These transition-stages are characterised by alterations of consistence and especially of colour. The red, hepatised lung gradually becomes paler, assumes a brownish-red, then a greyish-red or grey, and finally a yellowish colour, and thus presents the condition to which the term *grey hepatisation* has been appropriately given. We can recognise this coloration externally, but far better on examining a cut surface; and we can perceive that, in many cases, the tint is not monotonous, but that the black pulmonary tissue is more or less uniformly sprinkled over the greyish-red, grey, or yellowish-grey ground, which is also marked by the white projecting cut vessels, so that the whole presents a granite-like appearance.

The granular texture is still present, and even becomes decidedly more distinct at the commencement of the third stage, especially when the progress of the disease has been rapid and tumultuous; the consistence diminishes and the decoloration increases the nearer the disease approaches to the third stage; although the lung feels tolerably firm, it remains pitted after pressure, and is yielding and easily torn, and a greyish-red, very turbid, flocculent, viscid fluid exudes from its cut or torn surface.

If we examine the granulations in these transition-stages, we perceive that they have become more marked, larger, and more independent of the surrounding structures; and that they can be more easily separated and removed, as they only adhere loosely by a glutinous substance to the walls of the cells.

Third Stage, Purulent Infiltration.—At its commencement the change of colour of the hepatised tissue to a yellow tint (to which we have already adverted) becomes more or less uniform, the granular texture very rapidly disappears, and is succeeded by a purulent infiltration of the parenchyma. The lung then becomes heavy; any pressure on it forms and leaves a distinct pit; the cut surface is yellow or straw-coloured, with interspersed spots of black pulmonary tissue, and effuses a large quantity of a very viscid, purulent fluid of the same colour as the surface, and of a sickly odour; the parenchyma is extremely yielding, gives way on the slightest pressure, so that, if not carefully handled, cavities are easily formed in it, which are the more likely to be taken for abscesses, as they actually are very similar to fresh accumulations of pus. The granular texture has now altogether disappeared, and, on removing the pus from a piece of lung by careful pressure and washing, we perceive that its substance has again assumed its spongy, cellular tissue.

The bronchi present several changes, especially in their final ramifications; in the first stage their mucous membrane is reddened and swollen, subsequently however it becomes paler; and they almost always contain first a reddish, and afterwards a whitish, purulent, fluid exudation. The vessels are frequently clogged by exudations of this nature.

These are the three stages through which well-marked cases of acute pneumonia run; the last is the ordinary and natural mode of termination, and is frequently, although by no means necessarily, fatal, for, partly by expectoration and partly by resorption of the pus, the lung may return to its normal condition. There is no other and earlier stage than that which we have described as the stage of stasis, for the condition described as such by Stokes is in no respect inflammatory. The bright-red colour of the lungs or of portions of them, which Stokes regards as the earliest stage of inflammation, and attributes to arterial injection, is:—

(a.) Always dependent on anæmia, which is frequently very highly developed.

(b.) The lungs, or the affected parts of them, are puffed up, but are devoid of turgor and resistance in consequence of their capillary vessels not being duly filled; they collapse readily, and not a trace of a swelling of the tissue remains.

(c.) This condition always occurs when, in consequence of paralysis of the heart or of excessive thickness of the blood, the capillaries of the lungs can no longer be injected, and the little blood occurring in them is repeatedly exposed to the chemical influence of the atmospheric oxygen by the inspirations during the death-struggle. In this way we observe this condition either distributed over large portions of the lungs, or confined to small spots of lungs otherwise healthy, or associated with hyperæmia and stasis in many cases of asphyxia in newborn children and adults, in consequence of rapidly exhausting diarrhœas, of Asiatic cholera, after extensive burns of the general integument, &c.

Before entering into any further discussions, it will be most expedient that we should add to the above sketch the conclusions regarding the seat and nature of the pneumonic process, at which we arrive from an accurate anatomical investigation after death, and a review of the physical phenomena during life. These conclusions will not only find an influential application in what is to follow, but will also receive corroboration from it.

In relation to the first point, we have already stated, in our remarks on the formation of the granular texture of the hepatised lung, that the granulations are formed by the inflammatory product deposited in the cavity of the air-cells. Their formation, or, in other words, the exudation, is preceded by the secretion of a viscid, tough, reddish-brown fluid in the cavities of the cells, which gives rise to the crepitation well known to auscultators: as the stage of hepatisation advances this fluid disappears, and the air-cells become filled with plastic exudation. The granulations are roundish, and at first of a dark-red colour, hardish, and fragile; they appear to have uniformly coalesced with the swollen, dark-red walls of the cells, from which it is difficult to isolate and extract them. The inflammatory turgor and the redness of the tissue become then moderated; the

granulations become paler, of a greyish-red, and finally a greyish-yellow tint, while they appear less dense in structure, and become somewhat swollen. The secretion of a glutinous mucus is established around their circumference, which loosens their connection with the cell-wall, thus rendering themselves and the swelling more obvious: they appear surrounded by a light reddish cell-wall, and their distinctness is proportional to its paleness. Finally, they break down into a purulent fluid, mixed with this glutinous, mucous secretion. Hence the *seat* of the pneumonic process is on the walls of the air-cells—that is to say, on the *pulmonary mucous membrane*, and its product is deposited in the cavities of the air-cells; from this period—that is to say, from the stage of red hepatisation—the process consists in a metamorphosis tending to the fusion and breaking down of the exudation, under the influence of an inflammatory process, which is now declining in intensity. These conclusions are further strengthened by the following considerations:

(a.) If the granulations were regarded as swollen, and consequently obliterated, air-cells, they could neither exhibit the above anatomical relations, nor could they present the metamorphoses which, regarding them as inflammatory products, we have represented that they undergo; that is to say, if we take an unbiassed view of the subject.

(b.) Even the greatest swelling of the air-cells could not modify the volume of the hepatised lung, while our theory perfectly explains this phenomenon.

(c.) If the third stage, or that of purulent infiltration, were a suppuration of the interstitial tissue, a recovery from it without abscess and solution of continuity could not be possible; whereas it takes place by partial expectoration and partial absorption of the dissolved exudation, without any ulcerous destruction of tissue, in which case anatomical investigation shows, that, in purulent infiltration of the lungs, the texture is altogether undestroyed, and of a spongy, cellular nature.

(d.) Finally, the same process, as a general rule, extends to the terminal ramifications of the inflamed lung.

Even from what has been already said, it appears that, in relation to its anatomical elements, we may regard pneumonia

as a *croupous process on the pulmonary mucous membrane*, or, in other words, as a *parenchymatous croup*. It exhibits, even within the limited circle of its anatomical relations, a perfect identity with the croupous process on other mucous membranes; we shall, however, subsequently develop this view in a more extended and general manner.

We very often find the three stages coexisting, and can observe all the transition-stages passing into one another. Purulent infiltration and grey hepatisation generally predominate in the central and inferior part of the inflamed lobe; there is greyish-red or red hepatisation towards the periphery; above this there is inflammatory engorgement; while, finally, simple stasis, and very frequently acute œdema in different stages, are present in the adjacent tissue.

Pneumonia may prove fatal in any of these stages; it may also retrograde from each to the normal condition. Besides the above-described termination in purulent fusion of the inflammatory product (purulent infiltration), it may, in rare cases, give rise to abscess or induration, or may end in other ways, which can be more suitably noticed in a future part of the work.

If the pneumonia has reached the third stage, and is proceeding towards a cure, we observe the following phenomena:—The purulent fluid is gradually removed, and an exhalation of serum commences from the pulmonary mucous membrane; the pus which still remains is gradually rendered thinner by this admixture, and is finally converted into a flocculent, turbid serosity, which becomes mixed with air-bubbles as soon as the air again begins to penetrate. The parenchyma, at the same time, becomes paler, and of a greyish-yellow tint, and retains this colour for a considerable time; it crepitates less distinctly than in the normal state, is softer and moister, is more or less œdematous, and easily torn.

The lung can also retrograde from the second stage, that of hepatisation, to the normal state, without the purulent liquescence of the exudation. This process is undoubtedly one of the most difficult which the healing powers of nature can accomplish; for it always takes place somewhat slowly, and undoubtedly the more so in proportion, on the one hand, to the plasticity of the product, and, on the other, to the ex-

haustion following the effusion, whether the exhaustion be dependent on the disease, or be induced by the activity of the treatment. The granulations, together with the tissue, gradually become paler, and a serous fluid, which is secreted in the cells, seems by degrees to cause a fusion of the granulations, layer by layer. The tissue still retains a granular character, but the granulations always become smaller, of a pale red or reddish-grey colour, and are bathed in a serous fluid, which is mixed with tolerably consistent, pale reddish or whitish flocculi, and which gradually becomes frothy from the entrance of air. When the granulations are thus finally melted down, the parenchyma remains for some time in a state of serous infiltration, and is redder, firmer, and more resistant than in the normal state, owing, apparently, to a still existing infarctus of the walls of the air-cells and of the interstitial tissue. This retrograde process does not go on with equal or uniform rapidity at all parts; and we can often confirm our diagnosis by finding dense and still hepatised patches in tissue which has more or less returned to the normal state.

Finally, pneumonia retrogrades from the first stage—that of inflammatory stasis—to the normal condition; this is very frequently the case when those favorable influences are present which it is the great object of the healing art to induce. The inflammatory stasis, after it has deposited a moderate infiltration of turbid serous fluid, is converted into simple stasis, and after this is resolved, the tissue again becomes normal, but remains for some time the seat of hyperæmia which may easily relapse into inflammatory stasis.

Pulmonary Abscess.—We have already described the termination of pneumonia in purulent infiltration, that is to say, in purulent solution of the inflammatory product, which occurs without any separation of continuity or ulcerous destruction. The reverse takes place when accumulations of pus are formed in the lung. This termination of pneumonia is extremely rare; but this rarity need not excite our wonder, nor do we require the explanation attempted by Laennec, if we adhere to our view of the pneumonic process. The conditions giving rise to the formation of pulmonary abscess and the mode in which it is formed are, however, little known. Of all the theories which have been advanced, that is most conformable with the nature

of the pneumonic process which regards it as a consequence of a peculiar character of the inflammatory process, causing the pulmonary mucous membrane, which has been deprived of its epithelial investment, and the other tissues entering into the composition of the parenchyma, to become disintegrated and to suppurate,—a process analogous to that which occurs in many cases of true croup of the mucous membrane, and still more so to other exudative processes occurring on the same structure.

A recently formed, fresh pulmonary abscess presents the appearance of a cavern of irregular form filled with pus formed from the disintegration of the lung and surrounded by softened parenchyma infiltrated with pus, and in some places hanging in shreds. It is perfectly similar to those rents which may be produced by pressure, when we are carelessly handling a lung in the stage of purulent infiltration, or on attempting to separate it from adhesions to the costal wall, and which we have already warned our readers against mistaking for pulmonary abscess.

The abscess either enlarges in the same way in which it originated, by the continued solution of the inflammatory product and of the tissue of its walls, or else by the confluence of other neighbouring abscesses. As a general rule the suppuration extends over the whole of the inflamed portion of the lung, and hence the abscesses consequent on lobar inflammation (of which we have already spoken) are always very considerable. According to their size we observe one or more bronchial tubes opening into them with transverse or oblique mouths, and their tissues also become the seat of purulent solution. These abscesses represent the true but very rare *ulcerous pulmonary phthisis* which is based on inflammation.

It proves fatal either by the supervention of fresh pneumonia around it, or of pleuritis, or by the absorption of pus into the blood, with the symptoms of pyæmia and hectic fever. In rare cases it perforates the pulmonary pleura, and causes suppuration of the adjacent tissues, after having given rise to pleuritis and adhesion of the lung to the walls of the chest. Finally, in some very rare cases, it opens freely into the thorax before pleuritis and adhesion of the lung to the walls of the chest have been established; a general or circumscribed pleuritis then follows. If any of the bronchial tubes open into the

abscess there will also be pneumothorax, and it may happen that the pleuritic effusion will be ejected through the air-passages,—a phenomenon which, however, occurs much more frequently as a consequence of a reverse succession of the processes, namely, from primary pleurisy and consecutive corrosion and suppuration of the pleura. (See Pleuritis.) Finally, pulmonary gangrene sometimes arises in its vicinity, and the purulent solution of the tissue is converted into gangrenous ichor.

When the abscess has existed for a long time, its inner wall appears smooth, and its form is as nearly as possible round, and in the surrounding parts a secondary, interstitial inflammation may be observed, in consequence of which the parenchyma becomes converted into a cellulo-fibrous tissue, which surrounds the cavity of the abscess, and isolates it from the remainder of the pulmonary tissue. When an abscess is large, its perfect closure is very difficult; the process by which this is effected is by agglutination of its walls, which causes the obliteration of the bronchi entering into it; when the abscess has been a very large one, there is a depression of the thorax over it; and when its position is near the surface of the lungs, there is a puckered cicatrix left.

A pulmonary abscess may be confounded with a tuberculous vomica, and with certain accumulations of pus, which are developed from a secondary inflammation of the capillaries of the pulmonary tissue, of which we shall speak presently, and also with saccular dilatation of the bronchi. The diagnosis may be established from a comparative view of the positive signs attending each of their conditions.

Induration.—There are certain conditions under which hepatisation does not pass into a state of purulent solution, but into induration. The red inflammatory product becomes of a greyish red tint, and finally grey, but instead of becoming dissolved, it becomes compact and indurated. This is what has been termed *indurated hepatisation*, a condition which has sometimes, but incorrectly, been regarded as chronic pneumonia. The lung is compact, but fragile and pale, and has lost some of the increased size which it had attained during the stage of red hepatisation; it still, however, retains its granular texture, which even becomes more obvious, in consequence of the

granulations becoming more marked owing to their increased density, although they are somewhat smaller.

This condition may exist for a long time, and is always followed by cachexia, and especially by dropsical symptoms, and it often proves fatal; or the induration may be gradually resolved, or merge into obliteration of the air-cells and atrophy of the tissue.

The curative process in indurated hepatisation is somewhat analogous to the resolution of pneumonia in the second stage, for an exhalation of serous fluid takes place from the inner wall of the air-cells and acts as a menstruum, which gradually corrodes and absorbs the indurated granulations. As the granulations become smaller it becomes turbid and flocculent, and when the pulmonary cells are again permeable to air, it gradually assumes a frothy appearance.

In other cases the air-cells contract over the granulations, coalesce with them round their circumference, and become obliterated, their tissue being changed into a cellulo-fibrous structure, in which, from the similarity of their organisation, the granulations are most probably also merged. Unless a serous effusion occupy the empty space, this termination causes a depression of the thorax, or bronchial dilatation, or both simultaneously; and it appears to be on the whole less frequently the result of the croupous pneumonia which has already been described than of an insidious inflammation of the interstitial tissue,—interstitial pneumonia.

The above is a sketch of croupous pneumonia in general, when it occurs as a *primary* disease; but it also very frequently occurs as a *secondary* process. It most commonly runs an *acute* course, usually passing through its different stages in from two to three weeks, and in extremely rapid cases even in three or four days; these are, however, of rare occurrence. Sometimes, on the other hand, it runs a *chronic* course, being either nearly uniformly prolonged in all its stages, or one or other of them being especially protracted. It presents, however, no special relations essentially different from the sketch we have already given; for, like the acute form, it usually ends in purulent infiltration, and rarely in abscess or induration; and it is totally different from the affection which we commonly find described in pathological treatises as chronic inflammation of the lungs,

and with which we shall become acquainted when treating of inflammation of the interstitial tissue.

We observe variations in regard to the original *extension* of pneumonia, which are of importance chiefly in consequence of their connection with the inner nature of the disease.

Pneumonia, according to its variety, attacks, as we have already described, the whole of one of the larger divisions of the lung, that is to say, a whole lobe, or a great part of one, and it is then termed *lobar*. It often attacks a whole lobe and extends to the adjacent ones, and does not prove fatal till at length the remaining healthy lobes begin to be affected. It usually appears in this form as a primary affection; its most common seat is in the lower lobes, and the right lung is more frequently attacked than the left; both these rules present, however, many exceptions.

Or it attacks only smaller portions of the lungs, a number of individual lobules or single aggregations of lobules, between which we find the parenchyma in a comparatively normal state. It is then termed *lobular pneumonia*; it must be distinguished from the lobular hepatisations which are produced by irregularity in the progress of a lobar pneumonia in the individual lobules, while the rest of the parenchyma remains in a state of inflammatory engorgement.

Or, finally, the seat of pneumonia is confined to single air-cells; we then have what is termed *vesicular pneumonia*. The disease passes through the stages of inflammatory engorgement, of hepatisation, and of purulent infiltration in a single air-cell, or it causes induration, and, finally, obliteration of it. The indurated hepatisation of single air-cells is undoubtedly the same condition that has been described by writers on pathological anatomy, as *Bayle's pulmonary granulations*, and regarding whose nature there has been much unnecessary dispute. It is undoubtedly the result of inflammation, and so far Andral is correct in his view; but inasmuch as the inflammatory product, under certain conditions, assumes the character of tuberculous matter, it may also be regarded as partaking of the nature of tubercle (Laennec and Louis). It represents, as we shall presently show, the tuberculous infiltration of single air-cells.

Lobular and vesicular pneumonias are usually secondary processes.

It is very important that we should understand the differences presented by the inflammatory product in regard to its plasticity, inasmuch as they are most intimately associated with the condition of the blood (the general disease). Instead of the plastic, hepatised product, we meet under various conditions with serous, flocculent, and turbid, or gelatinous and glutinous, or sero-purulent, or even ichorous infiltrations, which, in consequence of their deficiency in coagulable matters, can never give rise to a granular texture of the parenchyma (hepatisation). The lung adjacent to the infiltration is dense and spleen-like, and in addition to the other marks of the inflammatory stasis, is generally discoloured, somewhat resistant to the touch, but on closer examination is found to be yielding, and is easily torn. Primary acute pneumonia usually deposits a plastic, hepatising product, which goes through the metamorphoses which have already been described; while, on the other hand, the last-named products are often the result of sluggish, asthenic (hypostatic) inflammations, and even more frequently of secondary pneumonic processes; they represent secondary exudative processes which not unfrequently degenerate into gangrene.

One of these pneumonic infiltrations, namely the *gelatinous*, must here be especially noticed. It is altogether different from the condition to which Laennec applied the denomination of gelatinous tuberculous infiltration, and which Andral, without hesitation, put down as the product of inflammation, and which we regard as the product of an inflammation of the interstitial tissue (see p. 90). In place of the plastic hepatising product, the air-cells are found to contain a gelatinous, viscid fluid, sometimes almost resembling frogs' spawn, and of a greyish, greyish-yellow, greyish-red, or brownish-red colour, and either clear and transparent, or flocculent and turbid, while the parenchyma is of a pale red tint, or more frequently of a reddish-brown colour, and is easily torn. The pneumonia which deposits this non-plastic product is chiefly observed around pulmonary tubercles, and especially around infiltrated tubercles and hepatisations which are undergoing metamorphosis into tuberculous infiltration; it is developed towards the end of the disease, and sometimes involves all the parenchyma which had remained free from tubercle and tuberculous infiltration; moreover we sometimes observe it in the vicinity of extensive

hepatisation, especially on the border of a hepatised lung in which emphysema has been developed, and which is impervious to a dense injection. Finally, it occurs whenever there is a deficiency of plastic matter for the deposition of a coagulable, hepatising product, either from some primary cause or in consequence of too profuse previous exudations.

Finally, we must here offer a few remarks on certain metamorphoses which constitute a hitherto undescribed termination of pneumonia, and which the plastic (fibrinous) hepatising product of inflammation undergoes in consequence of an inherent peculiar constitution depending on a general dyscrasia. These are its very frequent conversion into tubercle in the form of *tuberculous infiltration* or of *infiltrated tubercle*, and its very rare transformation (organisation) into medullary cancer, as *cancerous infiltration* or *infiltrated cancer of the lung*, to which we shall again return in our remarks on tuberculosis and cancer of the lungs.

There is a peculiar form of pneumonia to which the term *hypostatic* has been given by Piorry, and which is developed from the passive stasis which occurs in the most dependent parts of the lung, and to which the term *pulmonary hypostasis* has been assigned (see p. 64). It presents the thorough stamp of asthenic inflammation; for it is usually inert in its course, and lingers for a prolonged time in the stage of stasis, the parenchyma being of a dark livid colour, and gradually developing from isolated spots a lax, soft, livid-brown hepatisation, which may either be general or limited to separate foci, while a considerable portion, and occasionally even the whole, may become the seat of an inflammatory product in the form of a sero-purulent or gelatino-purulent infiltration without a trace of hepatisation. It constitutes the foundation of most of what are called latent inflammations of the lungs.

Primary pneumonia especially attacks vigorous adults, although delicate persons are also liable to this disease, and, indeed, not unfrequently seem decidedly predisposed to it; up to advanced age it is generally lobar, attacking at the least the whole of a lobe, and depositing a plastic, hepatising product in it. It further occurs in children, and even in new-born infants, presenting in this case several peculiarities; the granular texture of the hepatised lung is generally only indis-

tinctly seen, owing most probably to the density of the organ and the smallness of its cells; moreover the termination in abscess is relatively more frequent in children than in adults, and the lobular form is more frequently met with at this early age, although the simple catarrhal pneumonia is often mistaken for it. It arises in consequence of the influence of a peculiar atmospheric condition which predisposes to inflammation accompanied with abundant plastic exudations, and it may then be excited by many even very trivial causes; and in this point of view a notice of the combinations into which the primary pneumonic process enters is of importance, since they proceed from a common primary cause, namely, a peculiar, spontaneous, morbid change in the blood.

One of the constant symptoms is the sympathetic affection of the visceral surface of the pleura of the inflamed lobe, in the form of a thin plastic exudation investing it.

The anomalous condition of the blood which occurs in the pneumonic process, as well as in the other primary exudative processes, is a subject of much importance, since in this affection the change occurs in the most marked form and in the highest degree. In consequence of this circumstance we always find fibrinous coagula in the cavities of the heart as well as in the large vessels and their branches, and not unfrequently in those ramifications of the pulmonary artery which supply the inflamed lobe; they are distinguished by their yellowish and greenish colour, by their firmness, by a more or less decided metamorphosis into pus in their interior, by their similarity to the exudations on membranous expansions, and by their being woven among the trabeculae of the heart; and their partial coalescence with the endocardium and the inner membrane of the vessels, together with an obvious appearance of a secondary irritation in them, combine to show that they, at least in part, originated during life.

Pneumonia, if we except the pleurisy which co-exists with it, very frequently occurs as an independent disease (an exudative process) upon an extensive surface of mucous membrane, and may become more widely diffused in the lobar form, although it may, on the other hand, often be combined with similar processes upon other structures. Of these processes croup in the final ramifications of the bronchi is far the most common

(Lobstein), and is indicated by the presence of creamy, purulent, dissolving coagula in them. In children it occurs in combination with croup on the tracheal and other mucous membranes, and with exudations on serous membranes, as pleuritis, pericarditis, meningitis, &c.

Much interest attaches to the combination of pneumonia with secondary inflammations of the lining membrane of the blood-vessels, such as arise either from spontaneous coagulation of the fibrin in high degrees of hæmitis, and its becoming dissolved into pus, or, above all, such as occur in inflammations of the spleen terminating in ulcerous splenic phthisis.

Primary pneumonia proves fatal by inducing paralysis of the lungs; also from the supervention of pulmonary œdema or of other complications, from the high degree of blood-disease and the occurrence of spontaneous coagulations in the heart and vessels, and from acute softening of the stomach and œsophagus.

Secondary pneumonia is frequently developed as a result of inflammations in other organs, when they cause the blood to assume a consecutive disease similar to the spontaneous affection which we have already noticed; it frequently also accompanies specific processes which in their nature are allied to the exudative, and hence it especially occurs in the acute exanthemata. In both these cases the pneumonia is usually *lobar*. Finally, secondary pneumonia may occur as a metastasis towards the termination of various forms of acute dyscrasia of the blood, which, in their course, degenerate into a croupous diathesis; amongst these we must place many exanthematous, and the typhous and tuberculous processes; the pneumonia in these cases is generally *lobular* and may even be *vesicular*. Under this class we may also place many of the so-called latent, symptomatic, and, as has been already remarked, the metastatic inflammations. They are combined, especially under the circumstances which we have just mentioned, with exudative (croupous) processes of various degrees of plasticity on other mucous and serous membranes.

From all that has been stated, the croupous nature of the pneumonic process in general is sufficiently clear; being always based either on a peculiar primary (spontaneous) or on a secondary disease of the blood. There can be no doubt that this condition constitutes the basis both of secondary pneumonia and of the other metastatic croupous processes so frequently

combined with it, and it may also, in all essential points, be looked upon as the foundation of primary pneumonia and other primary croupous processes on the mucous membrane of the mouth, throat, and respiratory organs.

But as there are variations in the individual peculiarities, the age, and the external influences, under which croup of the mouth and pharynx, tracheal and bronchial croup, and, finally, croupous pneumonia, are developed, so also may the diseased condition of the blood vary in these affections, although probably only in a slight degree; and pneumonia, if we consider it as pulmonary croup, and if we take into consideration the plasticity of the exudation, may be regarded as occupying, in adult life, the same place which in earlier life is held by pharyngeal and tracheal croup; while bronchial croup, especially in adults, forms the transition between the two latter varieties and pulmonary croup (croupous pneumonia). We now proceed to the consideration of *typhous pneumonia*, in consequence of the similarity of its anatomical relations to those of croupous pneumonia.

Typhous Pneumonia (Pneumotyphus).—The pneumonic process is very frequently associated with the typhous; but its relation to the latter, and especially to the local typhous process on the mucous membrane of the ileum, is not always the same, and hence its importance varies.

In all cases of typhus, and especially when there is well-marked ileo-typhus, there is hypostasis in the lower lobes; and this not unfrequently becomes developed into pneumonia, which deposits a gelatinous, glutinous, soft product, similar to the typhous, bronchial and intestinal secretion, and corresponding to the existing typhous dyscrasia. It is the result of an adynamic state of the system, and bears no further definite relation to the typhous process, which is seated on the intestinal mucous membrane.

A more intimate relation, however, exists when the typhous process has been originally localised in the pulmonary mucous membrane to the exclusion of other structures, especially the intestinal mucous membrane, namely, in *primary pneumotyphus*; and when, in consequence of its absolute intensity or its relatively imperfect localisation on the intestinal mucous membrane, it also appears in the lungs, and completes the local process on the intestinal mucous membrane, as *secondary pneumotyphus*.

Primary Pneumotypus is a (croupous) lobar pneumonia characterised by the livid and almost violet colour of the parenchyma during the first stage, and by a dirty brownish-red or chocolate-coloured, very yielding inflammatory product (hepatisation), which soon breaks down when there is great disease of the blood and extreme absence of plasticity. It seems to be always combined with bronchial typhus, and the bronchial glands exhibit the characteristic relations of this affection. It exists either without or with only a slightly marked secondary affection of the intestinal mucous membrane, and, in association with bronchial typhus, doubtless constitutes most, if not all, of those cases of typhus,—and especially exanthematous typhus,—which run their course without any local intestinal affection. Like genuine pneumonia it is usually combined with a pleurisy yielding a similar product.

Secondary Pneumotypus in its genuine form consists of an imperfectly developed local typhous process on the intestinal mucous membrane, has the same anatomical characters, but does not, as a general rule, attain the same degree of intensity and extent, which is presented by the primary form, when it meets with no obstruction to its original local development. It also enters into the same combinations, and is very frequently associated with genuine secondary laryngotyphus.

Secondary pneumotyphus occurs, however, much more frequently in a *degenerate* form, as a local expression of the degeneration of the collective typhous process, and, indeed, in the form of a lobular or vesicular pneumonia yielding a purulent and diffuent product, and very frequently associated with a form of laryngotyphus which has degenerated into croup; or it occurs in the form of purulent, diffuent deposits in the interstitial tissue, with inflammation of the capillaries of the lungs (purulent metastasis); or finally in the form of pulmonary gangrene.

2. *Catarrhal Pneumonia*.—Catarrhal pneumonia has hitherto received little attention, in consequence of its resemblance to the croupous variety, for which it may easily be mistaken, and on account of its rare occurrence in adult life. It is, however, comparatively common in children, in consequence of the large amount of undeveloped granular texture that is observed in hepatisation of their lungs.

It constitutes the first of the series of catarrhal affections to which the respiratory mucous membrane is exposed during childhood, and is succeeded in later years by bronchial and, finally, by tracheal catarrh; in this respect it is the opposite to croup, which begins in childhood as pharyngeal and tracheal croup, and which, in the form of pulmonary croup, terminates the series of croupous inflammations in adults. Catarrhal pneumonia is always lobular, and associated with a catarrhal affection of the bronchial tubes pertaining to the diseased lobules; it is frequently found in the various catarrhal affections of children, especially in pertussis and catarrhus suffocativus. Its usual position is in the superficial lobules, of which a very considerable number are often affected. They present, for the most part, a bluish-red tint, and are dense and somewhat firm; the walls of their air-cells are swollen, till no internal cavity remains, or if the swelling be less considerable, their cavities contain a watery, mucous, and slightly frothy secretion; there is no trace of granular structure. As the lung-substance in the immediate vicinity of the diseased lobules is usually emphysematous and pale, they appear to be a little depressed below the level of the surrounding lung, if they are situated near the surface, and they may be further recognised by their dark colour.

This disease frequently becomes fatal by the supervention of pulmonary œdema and paralysis, or by the stasis induced in the heart by the emphysema.

3. *Inflammation of the Interstitial Tissue of the Lungs.—Interstitial Pneumonia.*—This is a disease whose anatomical characters are not properly recognised in pathological treatises, for it is commonly described as *chronic inflammation* of the lungs consequent on ordinary croupous pneumonia, without any reference to its seat in a special tissue. The seat of this inflammation is the interstitial cellular tissue of the lungs, although the walls of the air-cells are often also implicated, in which case the pneumonia sometimes assumes the croupous form.

Its course is, as a general rule, chronic, and it is only very rarely that we have the opportunity of studying it, except in its final effects. So far as we can conclude from our few observations, it appears to commence in the tissue lying in the interstices of the pulmonary lobules and between the smaller groups of air-cells, which, if too much black lung-substance

be not present, becomes of a pale red colour, and is swollen by albuminous infiltration, while the air-cells are either pale and more or less compressed in proportion to the swelling; or, if they are involved in the inflammation, they appear reddened and, in accordance with what has been already stated, sometimes finely granular. In the progress of time the infiltration within the interstitial tissue becomes organised and coalesces with the latter, so as to form a dense cellulo-fibrous substance, which compresses and obliterates the air-cells, and finally converts them into a similar cellular tissue. We then find either whitish, hard stripes, which not unfrequently grate under the knife, or irregular masses interwoven in the lung-substance.

This is the ordinary metamorphosis consequent on chronic pneumonia; in some cases, however, it may terminate in supuration which isolates the individual lobules; and some pulmonary abscesses probably originate in this manner.

It is not very unfrequently a spontaneous affection, insidiously spreading from one lobule to another; it is commonly seated in the apices of the upper lobes, and, as we may infer from the co-existing cellular adhesions corresponding to their seat and distribution, it is frequently combined with circumscribed pleurisy.

The affected portions of lung become depressed, and draw down the surrounding parenchyma in the form of cicatrix-like folds, which may sometimes be observed on the apices of the lungs in cases where there is no trace of the pre-existence of tubercle. A further consequence of this process is a depression of the thorax at the corresponding spot, and, internally, a dilatation of the bronchial tubes.

More frequently, however, it is a consecutive affection, arising from reaction, and leading to the production of cyst-like formations around the seat of old apoplexies, abscesses, tuberculous caverns, gangrene, &c.; its products then resemble the tissue of which cicatrices are composed.

This tissue sometimes contains a considerable quantity of pigment (the black pigment of the lungs); it then presents blackish-grey stripes and spots, or else is uniformly of a blackish-blue tint.

g. Deposits in the Lungs. Metastatic Processes.—As a consequence of the absorption of a pseudoplastic process into the

living blood, or, more rarely, as a consequence of the spontaneous disease of that fluid, there is a process developed which is fully discussed in its general bearings under the head of "the diseases of the blood:" it, however, affects the lungs more frequently than any other organ, and usually occurs simultaneously at several circumscribed spots. It consists in the deposition of a fibrinous product in the lung-substance, or of a coagulation in its capillaries (phlebitis capillaris), either of which undergoes metamorphoses corresponding to the principle taken up into the blood.

As, on the one hand, the veins seem to be the seat in which deleterious substances are produced, or in which they are collected from without, and as, on the other, the whole of the venous blood passes through the lungs,—the principal organ in the process of hæmatisation,—it is easy to understand why it is that in general these deposits are most frequent and most abundant in the lungs.

As is generally the case in all parenchymatous organs, these deposits almost always occur in the superficial layers of the lungs. We find deposits of various dimensions, from the size of a millet-seed to that of a lentil, a pea, a bean, or even a nut, scattered through the tissue of the lung, and separated from one another by large patches of healthy tissue; the smaller they are, the more they resemble, in form, a roundish granulation, while, on the other hand, the larger they are, the more they lose the round form, and appear as irregular, angular, ramifying masses. Large deposits, when lying near the surface, and pressing upon the pulmonary pleura, like those occurring in the spleen, have a wedge-like shape, being thick externally, and growing small towards the interior. They are at first of a blackish-red or brownish-red colour, and firm although fragile, and can be distinguished by their sharply defined outline, and by their apparently homogeneous structure, from the surrounding tissue, which at the commencement is normal, or at most the seat of hyperæmia and œdematous infiltration; but subsequently, when the deposit begins its progressive metamorphoses, a reactive inflammation, in the form of croupous pneumonia and hepatisation, is set up in the lungs, and its extent is usually proportional to the size of the deposit.

The deposit subsequently becomes of a lighter colour, and undergoes one of the following metamorphoses:—

In one case (and this is what commonly occurs), the deposited mass becomes more or less decolorised, and dissolves into a cream-like, purulent, or ichorous fluid, which destroys the tissues. This process commences in the centre of the deposit, much as we observe to take place in secondary phlebitis of one of the larger veins, and we then find the above-named fluid enclosed within the outer remains of the deposit, around which a reactive inflammation is established. In the course of time these remains, and the adjacent tissue also, undergo a similar process of fusion, and the extent of this change is proportional to the destructive tendency of the product of the reactive inflammation. Moreover, this process is very often essentially of a septic nature, and is based on the absorption of gangrenous ichor, or, on the other hand, it often undergoes degeneration, and gives rise to gangrene of the surrounding tissues. These deposits are very frequently combined, from the first, with a secondary pleurisy of a croupous nature; sometimes, however, the latter occurs as a consecutive affection, arising from the inflammatory reaction that is set up around the superficial deposits, or as a purulent or ichorous abscess in the immediate vicinity of the pleura. In the latter case, we observe the abscesses as roundish, nodular, furuncular, yellow prominences, or if gangrenous destruction has occurred, as dirty-greenish or brownish collapsed spots, shining through the pulmonary pleura, which itself undergoes destruction from suppuration or gangrene, with or without perforation, and gives rise to general pleurisy.

In the other metamorphosis, which, however, is extremely rare, the deposit, without dissolving or undergoing any intermediate change, passes directly from its crude state into that of obsolescence, that is to say, it shrinks into a callous, greyish nodule, which is seated in a capsule of cellulo-fibrous tissue, and in the course of time becomes converted into an osseous concretion. Many of these peripheral deposits, after their conversion into concretions, have doubtless been mistaken for chalky tubercles. The more complicated retrograde process which is sometimes manifested in deposits in other parenchymatous organs, as for instance the spleen and the kidneys, namely, the cheesy disintegration of the product, and its subsequent conversion into chalky matter, may, as we should

presume from analogy, also occur in the lungs; but, in the whole course of our observations, we cannot recollect a single case in which it has occurred.

We have spoken in the first volume of the rarity of obsolete deposits in the lungs, and have also accounted for it.

We have already explained how these deposits become combined with pleurisy; they are also associated with similar deposits in other parenchymatous structures—as the spleen, kidneys, liver, brain, and thyroid gland, in the tissue of mucous membranes, especially that of the intestines, in the skin, the subcutaneous cellular (areolar) substance, and all interstitial cellular layers, and in the muscles; also in the exudative processes on mucous, serous, and synovial membranes, (as, for instance, metastases in the joints.)

They must be carefully distinguished from lobular pneumonia, for which they have sometimes been mistaken.

h. Gangrene of the Lungs is an affection of not unfrequent occurrence, and one which, as Laennec very correctly remarks, must not be regarded as the result of an excessively acute inflammation. We do not, however, intend to assert, that it cannot by any possibility occur in an inflamed lung, for under certain conditions hepatisation of a portion of the lung is unquestionably the most common complication.

We will first consider it in an anatomical point of view, and then proceed to notice the conditions under which it is developed.

There are two perfectly distinct forms of gangrene of the lungs, namely, *diffuse* gangrene and *circumscribed* gangrene or *gangrenous eschar*.

In *diffuse* gangrene, we find a portion of the lung presenting an abnormal greenish or brownish tint, filled with a similarly coloured, somewhat frothy, turbid serosity, soft, rotten, and readily breaking down into a pulpy, shaggy tissue. The whole evolves the characteristic odour of sphacelus. Towards the outer portion the discoloration, infiltration, and alteration of consistence are less marked, and finally become imperceptible; and there is no line of demarcation between the gangrenous and the adjacent tissue, which only differs from the normal state in being œdematous and anæmic. It corresponds to diffuse gangrene of the bronchial mucous membrane, with which it is almost

always associated. Upon the whole it is a rare affection; but when it does occur, it always attains a considerable extent, as it commonly attacks the whole of a lobe, or, at all events, its greater part. It especially attacks the upper lobes, when, in consequence of excessive activity, they have become the seat of emphysema and anæmia, the lower lobe being at the same time in a state of passive stasis. It is perhaps scarcely entitled to rank as an essentially independent affection, inasmuch as it is almost always associated with gangrenous eschar of the lungs; and hence it is the more readily developed from the contact of the ichorous, gaseous, and fluid products of the gangrenous eschar coming in contact with the bronchial and pulmonary mucous membrane, inasmuch as in all probability the disease extends from the bronchi to the lung-tissue. The above description of gangrene, as it occurs in the upper lobes, is sufficient to render this form intelligible, as well as to explain why there is no inflammatory reaction, and, consequently, no line of demarcation around the affected tissue.

As we have already remarked, it must be carefully distinguished from *softening of the lungs*.

Circumscribed or partial Gangrene of the lungs appears in the form of gangrenous eschar, and is incomparably more frequent than the former variety. We find the parenchyma, at some spot of varying size, converted into a blackish or brownish-green, hardish, but moist and tough eschar, which adheres to the surrounding tissue, evolves, in a very marked degree, the peculiar odour of sphacelus, and, as Laennec observes, is extremely similar to the eschar produced on the skin by nitrate of silver. It is sharply defined, and, as we shall presently show, the surrounding parenchyma may be in various conditions.

The eschar becomes gradually loosened from the surrounding tissue, and rests in an excavation corresponding to it in size and form; it may be described as a blackish-green plug, which superficially is soft, shaggy, moist, and bathed in an ichorous fluid, but, towards its centre, is of a denser structure. More frequently, however, the whole or the greater portion of the eschar softens and becomes dissolved into a greenish-brown, very fetid, ichorous pulp, mixed with rotten, shaggy fragments of tissue, and enclosed in a cavity whose walls are lined by a shaggy tissue infiltrated with ichor.

The size of the gangrenous portion, at its commencement, varies from that of a bean to that of a hen's egg, or may be even larger; it is most commonly not smaller than a hazle-nut or larger than a walnut. The form is on the whole irregular, with a tendency to roundness. It is much more commonly seen in the superficial than in the deep layers of the lung, and more frequently in the lower than the upper lobes.

These eschars may either occur singly, or several may be simultaneously present.

The number and size of the bronchial tubes attacked by the gangrenous destruction, are usually proportional to the size of the original gangrenous centre; these bronchial tubes constitute the passage through which the gangrenous exhalation and the eschar itself, in the respective form of an intolerably fetid atmosphere around the patient, and of gangrenous, ichorous sputa, make their escape. The gangrene proceeds outwards, and attacks the pulmonary pleura the more quickly the nearer it was originally seated to the surface of the lung. If the gangrenous eschar becomes detached, it falls into the cavity of the thorax, unless there are firm adhesions at the spot; or else it becomes dissolved, and the ichorous semi-solid matter is effused into the pleural sac, and gives rise to pleurisy with ichorous exudation, and to pneumothorax, since the fetid gas evolved from the gangrenous mass either collects alone in the thorax, or atmospheric air finds its way through the bronchial tubes which open into the abscess, and thus mixes with the aforesaid gas in the thorax. These superficial gangrenous caverns may be recognised at a glance, for at these spots the pleura is either converted into a blackish-green eschar, whose inner surface is shrivelled and hard; or, if the eschar has already dissolved, the pleura is of a blackish-green colour, rotten, and moist, and appears distended by the gas evolved from the abscess; or, finally, if the pulmonary pleura be ruptured at certain spots, or be perforated, or even perfectly destroyed, in consequence of spontaneous fusion, we shall observe the open, sunk cavern, either covered by the remains of the pleura, or thoroughly exposed, and more or less completely emptied.

A primary gangrenous abscess must be distinguished, when possible, from one that has undergone subsequent enlargement; very large abscesses are, as a general rule, not primary, but are

formed by the corroding action of circumscribed gangrene, and do not, as we shall presently show, present the distinct line of demarcation which is observable in primary abscesses.

The lung-substance surrounding the gangrenous abscess is sometimes normal, with the exception of a serous or sanguineo-serous infiltration; but when the gangrenous eschar dissolves, diffuse gangrene may be developed in it to a greater or less extent. More frequently, however, we see it in a state of reactive inflammation, varying in extent and character. Very often there is a simple stasis of an asthenic character; this gradually assumes an inflammatory type, which it retains for a long time, and then the stage of hepatisation slowly and imperceptibly ensues. From a want of energy in this process of reaction, the primary gangrenous abscess may extend in various directions, so as often to attain the size of a man's fist, or even of a child's head, while the surrounding tissue becomes more or less rapidly discoloured, without presenting any decided stratified appearance, and finally breaks down into a gangrenous, ichorous pulp. In this way the gangrene may extend outwards, until it reaches the pulmonary pleura, when it may give rise to the consequences which have been already enumerated; and indeed, if the lung be adherent, the costal pleura at the corresponding spot may be involved in the metamorphosis.

We often find a higher degree of inflammation set up in the surrounding tissue; it is in a state of decided hepatisation, which sometimes extends over the whole of the lobe which is affected by gangrene. The disease not unfrequently proves fatal through this excessive reaction.

The most important process, however, occurs in the layer of tissue immediately surrounding the cavern, and is obviously an effort of nature to promote a cure. The reaction here appears as an inflammation of the interstitial tissue of the lungs, which, together with the cavernous walls, undergoes suppuration, and thus effects the removal of the sphacelated tissue which was adhering to the walls of the abscess.

In this process we find that at first only single or isolated patches of tissue become gangrenous, and the pus which is secreted from the walls of the abscess is still mixed with ichor, and gangrenous fragments of tissue. As the process advances, however, suppuration predominates, and after the gangrenous

tissue has been ejected through the bronchi the cavern is converted into an ordinary suppurating abscess, whose inner wall is infiltrated with pus; externally, for a distance varying from three to six lines, the tissue is of a greyish-red colour and firm; and if croupous exudation in the air-cells be associated with the inflammation of the interstitial tissue, we observe a scarcely perceptible, very delicate granulation. If the suppuration in the inner stratum of the capsule now diminishes, the result of the whole process is a cavity, with whitish, cellulo-fibrous, callous walls, which sooner or later coalesce, leaving merely a cicatrix, like ordinary abscesses or tuberculous vomicae. In some rare cases circumscribed pulmonary gangrene undergoes a cure in this manner.

If the eschar breaks down, and dissolves very rapidly, and little or no reaction be developed in the surrounding parts, or if the primary cavern enlarge very quickly at the expense of the surrounding parts, the gangrenous destruction not unfrequently involves large, unobliterated blood-vessels, and gives rise to exhausting hæmorrhages into the cavern, the bronchial tubes, or even into the thoracic cavity, when the abscess has opened into the pleural sac.

Partial gangrene often arises in the perfectly healthy lungs of weak, decrepit, cachectic persons from general depressing influences, and is developed from a circumscribed passive stasis. Under similar circumstances, we find it associated with pneumonia in its various stages, with pulmonary abscess, with pulmonary tuberculosis and tuberculous vomicae, with bronchitis, especially when it is developed in the course of exanthematous diseases, both in adults and children, &c. Finally, it appears among the sequelæ of typhus, as a manifestation that the typhous process is spontaneously degenerating into a state of putrescence; or it may be produced by the absorption of gangrenous ichor from gangrene of different parts into the blood, in which case we have diffuent gangrenous deposits, or septic capillary phlebitis.

i. Softening.—Softening of the lung-tissue is of very rare occurrence; it is altogether distinct from pneumonia, and must not be confounded with Andral's ramollissement (red and grey hepatisation); like softening of the stomach, it is a peculiar spontaneous process, and appears under precisely the same

conditions as that affection; indeed, as a further proof of their identity, we may add that this disease is almost always combined with gastric softening.

In any part of the lungs we may find an undefined patch of a dirty brown or blackish colour, according to the state of the blood at the commencement of the process, and which is so very moist and soft that on the slightest pressure it breaks down into a pulp, which is mixed with a serous fluid and contains black flocculi of carbonised blood. The bronchial mucous membrane is found in the same state for some distance around the diseased spot.

In consequence of the considerable quantity of blood which is always contained in the lungs, there is a resemblance between softening of the pulmonary tissue and the black softening of the stomach, which proceeds from a disease of the blood itself. It may be easily mistaken for diffuse pulmonary gangrene; indeed in very intense cases the diagnosis must depend on the absence of the gangrenous odour, and on the lesser degree of discoloration.

k. Adventitious Products.

1. *Cysts* are of extremely rare occurrence in the lungs, which in this respect present a marked contrast with many of the other parenchymatous organs.

Simple Serous Cysts may, doubtless, occasionally be found in the lungs, but *sacs containing Acephalocysts* are of less rare occurrence. The rarity of the latter cysts in the lungs contrasts strongly with their frequency in the liver, and this is very important when we consider the frequency of pulmonary tuberculosis, for this, in addition to the inverse ratio of the frequency of these secondary products in other organs, especially in the liver, constitutes one of the most important objections to the theory that tubercle has an hydatid origin.

Hitherto only single sacs of acephalocysts appear to have been found in the pulmonary tissue; they have varied from the size of a pigeon's egg to that of a man's fist, and have occurred sometimes in the upper and sometimes in the lower lobes.

They are undoubtedly developed in the interstitial tissue of the lungs, and occasion, according to their size, more or less compression of the parenchyma, which is thus gradually converted into fibro-cellular tissue (obsolescence). The parent

sac is surrounded by and adherent to this tissue, and contains, in its interior, the acephalocysts, which vary in number and form, and either swim freely in a serous fluid or are attached to the walls.

It is important to recollect that in rare cases the parent sac may be destroyed by inflammation and consequent supuration, and a communication may thus be established between the cavity and the bronchi, through which the acephalocysts may be ejected, especially as in less rare instances acephalocysts are ejected from the liver by this complicated route.

The pulmonary sac containing acephalocysts often communicates with a similar sac in the liver.

Cysts containing other substances, as for instance cholesterin, with or without hair, are even rarer than cysts with serous contents.

2. *Anomalous Fibrous or Fibrocartilaginous Tissue* occurs—

a. As *callous condensation* arising from chronic inflammation of the interstitial tissue; it likewise occurs as *cicatrix-callus* around old abscesses, tuberculous cavities, apoplectic effusions, &c.

b. *Fibroid Tumours* are incomparably rarer. They never attain any considerable size, being seldom larger than a bean or a hazel-nut. They are either bluish-white, firm, elastic, very dense and flat bodies, or, as is far more frequently the case, they are of a pale yellow or dirty white colour, flabby, soft, and puckered, and resemble the structure of the mammary or salivary glands.

3. *Anomalous Osseous Substance* occurs not only in the bronchi (see p. 29) and bronchial glands, but, under various circumstances, in the lungs, especially in the form of ossification of anomalous fibrous tissue or of the chalky metamorphosis of an unorganised structure. To the first belong many either flat, or roundish and nodular, yellow, and generally very compact concretions, which are developed in and from all the forms of anomalous fibrous tissue, but especially in the callous stripes, capsules, and cicatrices; to the latter belong the chalky, whitish or greyish nodular, brittle, and even friable masses into which tubercle and tuberculous pus are, under certain conditions, metamorphosed.

4. *Black Pigment* is more frequently and abundantly deposited in the lungs and bronchial glands than in any other organ, except the mucous membrane of the intestinal tract. It occurs, with rare exceptions, in the lungs of all adults; and increases with advancing years. Hence it can properly be only regarded as a pathological appearance, either when it occurs in the earlier periods of life or in excessive quantity.

The pigment which occurs in the form of molecules is either deposited in a *free* state in the interstitial tissue and in the walls of the air-cells, or else it is *combined*, as a new formation, with some older deposit.

In the *first* case it is found according to the extent of its accumulation, in blackish-grey, blackish-blue, or ink-black points, or in patches, as if laid on with a brush; or if very abundantly present, it is diffused over the interstitial tissue in large ramifying streaks, which appear as islands in the cellular tissue under the pulmonary pleura, and are uniformly infiltrated, blackened, and as it were inked, and are thickened and tough. This thickening of the interstitial cellular tissue is important, since it impedes the development of the air-cells, and likewise gradually obliterates their vessels, and in this manner causes their atrophy. We must here especially notice a metamorphosis which not unfrequently occurs at the apices of the upper lobes, and is unassociated with any other anomaly; we refer to the deposition of large quantities of pigment which give a black colour to the tissue, and increase its firmness, its structure being either normal or presenting at some spots an irregular reticulated appearance in consequence of atrophy. *Senile atrophy* of the lungs is undoubtedly often induced by an excessive accumulation of pigment in the interstitial tissue. The deposit usually takes place through the whole lung, but is most abundant near the surface and in the upper third of the superior lobes. It is the result of slight irritative processes and of transient stases; the pigment is conveyed by absorption to the bronchial glands, and is thus deposited in them. It must be decided by further and more careful observations whether the large deposits of pigment which are so frequently noticed in the lungs of persons engaged in working both coal and coal dust, depend upon the actual absorption of these extraneous matters into the tissue, or whether, as we are more

inclined to believe, they are results of the continued irritation to which the pulmonary mucous membrane of such persons is necessarily subjected.

In the *second* case the pigment is the result of a chronic pneumonia, and we find it infiltrated in various quantities into an indurated and callous parenchyma. We meet with it in the vicinity of tuberculous deposits, especially of hæmorrhagic tubercle, and in certain cancerous deposits, especially in cancer melanodes.

5. *Tubercle.*—*Pulmonary tuberculosis*, which is the most frequent of all the tuberculoses, is one of the most common and likewise the most fatal of the diseases of the lungs.

For general information on tubercle and tuberculosis we must refer to what has been stated in the First Volume; we shall here endeavour to apply those general principles to a special case, and at the same time shall attempt to elucidate certain points which, from presenting some peculiarity, require a fuller notice.

Tubercle does not primarily occur in the lungs in the numerous forms which have been described, but only in *two* forms, which are most essentially connected, both in relation to its mode of formation and its seat. To these two forms we apply the respective terms of *interstitial tuberculous granulation* and *infiltrated tubercle* or *tuberculous infiltration*.

a. *Interstitial Tuberculous Granulations* occur in the pulmonary texture in the form of roundish, originally grey, semi-transparent bodies, varying from the size of a hemp or millet-seed to that of a barleycorn; these minute bodies either occur singly and in an isolated state, or several are collected into a group, or finally they may coalesce and form a large continuous mass. They are seated, as is shown both by special anatomical investigations and by numerous analogies, in the interstitial tissue between the smallest lobules and the air-cells, and on the walls of the cells themselves; that is to say they are altogether external to the cell-cavities; but by pressure on the cell-wall they sometimes induce a corresponding internal prominence, or if they be of larger size they exert such pressure on the walls that in every group or confluent mass of tubercles we find a number of cells more or less completely obliterated. It is the result of a *chronic* or *acute tuberculous*

process, which is accompanied by local congestion or hyperæmia. We have now sufficiently indicated this form of tubercle, but we shall hereafter return more fully to it. For the sake of brevity we shall always name it *tubercle*, or *tuberculous granulation*, and it must be carefully distinguished from the second form, which we shall invariably term *tuberculous infiltration*, or *infiltrated tubercle*.

b. Infiltrated Tubercle, unlike interstitial tubercle, is actually deposited in the cavities of the air-cells. It arises from a more or less extensive croupous pneumonia whose products, under the influence of a tuberculous infiltration, become variously discoloured, and converted into yellow tubercle, instead of being absorbed or dissolving into pus. Hence tuberculous infiltration presents the form of *hepatisation*, or more strictly speaking is hepatisation, induced by a tuberculous product. The pneumonic product, which was at first red and granular, gradually becomes of a paler and greyish red colour with a tinge of yellow, and is dry and fragile; it finally becomes yellow, moist, of a soft, fatty, cheesy character, and sooner or later becomes disintegrated into tuberculous pus. The granular texture, in the mean time, gradually disappears, whilst the tissue forming the air-cells becomes tuberculous, and the diseased portion of lung appears to be actually changed into a connected fatty-cheesy tuberculous mass,—a condition which Lobstein doubtless observed, and mistook for *fatty metamorphosis* of the lung-substance.

This form of tuberculosis may attack *a whole lobe* uniformly, or even *a whole lung*, according to the extent of the local pneumonic process; it is, however, much more frequently confined to one or several larger or smaller separate portions of lung, and very often occurs as a lobular tuberculous infiltration, and in both these cases it is generally sharply defined; finally, it may occur as *vesicular tuberculous* infiltration, in which case it is the same thing as Bayle's *pulmonary granulations*, regarding which there has been much discussion.

It very often attacks the superficial parts of the lungs, as lobar and lobular infiltration, and may then be at once recognised by its external characters, by the pneumonic tendency, and the peculiar colour of the diseased portion.

It is always the result of a high degree of tuberculous

dyscrasia, and hence it only rarely occurs as primary tuberculosis, but is, as a general rule, associated with advanced stages of interstitial tubercle. It gives rise to a form of phthisis which is tumultuous and acute, is accompanied with repeated attacks of pneumonia, and is attended with much pain and distress.

It is especially frequent in young persons and children, and presents an analogy with bronchial tuberculosis, with one of the forms of tuberculosis of the intestinal mucous membrane, with the tuberculous metamorphosis of exudations on serous membranes, &c. It is always combined with a high degree of tuberculosis of the bronchial glands, and very often with tuberculosis of the intestinal mucous membrane.

These are the two principal forms of pulmonary tuberculosis, and all other varieties of tubercle, such, for instance, as depend on physical peculiarities, however important they may individually be, are unimportant in reference to the local process, depending either on different modifications of the general disease or on mere changes in the tuberculous matter.

There are no organs excepting the spleen and serous membranes in which tubercles occur in such great numbers as in the lungs. They appear either as *separate granulations* or several of them are accumulated into *one group*. In the first case each granulation is isolated from the others by an extent of lung-tissue proportional to the number of the tubercles. This takes place either in a comparatively uniform or in an irregular manner; the latter occurring when in one part of the lung we find a large number of tubercles with little intervening parenchyma, and in another a few tubercles interspersed among much healthy tissue. When the tubercles are present in large numbers they become pressed upon one another, and finally coalesce in the form of irregular masses, as may be especially observed in the apices of the lungs, where the disease is usually the most developed. In many of the more common cases we find an uniform increase in the number of tubercles, and a corresponding approximation of them to one another as we advance from the lower portions of the lungs towards their apices.

This accumulation of tubercles into irregular masses, such as occur in the apices of the lungs, which are the usual starting-

points of pulmonary tuberculosis, and occasionally at other spots, must be carefully distinguished from *the primary development of tubercle in tolerably regular groups*. Under certain local and general conditions which are not yet altogether understood, tubercles are originally deposited at different spots in groups of a roundish form, and of the size of a pea, a bean, or a hazelnut, or even larger, while around them there are usually other isolated tubercles in greater or lesser number. In extreme cases of this kind the tubercles are deposited around a central nucleus of pulmonary tissue, from which processes run into the tuberculous groups, dividing them into several compartments.

Pulmonary tubercles originally appear either (1) as the well-known grey, semi-transparent granulations of the size of a millet or hemp-seed, or in many cases of acute tuberculosis as still smaller-sized granules, which are clear, transparent and vesicular; or (2) in high degrees of tuberculous disease, especially when it is running an acute course, they are separated from the blood as yellow tubercle. On a cursory examination they appear almost or quite round; on closer investigation, however, we find that their outlines are not sharply defined, but that delicate prolongations extend from their surface into the surrounding tissue, which, according to their size, may enclose one, two, or more air-cells. These cells are most commonly obliterated, but not unfrequently appear dilated.

In examining the lungs we not unfrequently meet with extensive, roundish, or irregularly ramifying or lobulated tuberculous masses, which are produced either by the confluence of several tubercles which were originally in the same group, or by the subsequent deposition of tubercles in the same immediate neighbourhood. The tissue at these points is completely wasted away, so that nothing but the pigment remains, and the air-cells and extremities of the bronchial tubes are obliterated. These tuberculous masses must be distinguished as carefully as possible from tuberculous infiltration. The whole of the upper lobes are not unfrequently so thickly strewed with tubercles as to present the appearance of having degenerated into a tough, resistant, uniform tuberculous mass.

In the ordinary course of the disease the principal seat of the tubercles is in the upper third or apices of the superior

lobes; it is here that they are deposited first and in the greatest quantity, and that they first begin to undergo their ordinary changes. The apices of the lungs must therefore be regarded as the usual starting-point of tuberculosis, which gradually extends from thence to the lower portions of the lungs. Exceptions to this rule are, however, not unfrequent; we sometimes meet with tubercles in the apices and others far away from them, even in the lower lobes, or they may even occur in the latter portion while the upper parts are perfectly free from them. In this respect there is a contrast between pulmonary tuberculosis and pneumonia, at all events in a great majority of cases, for pneumonia most commonly commences in and starts from the lower lobes, while tuberculosis has its origin in the upper lobes, and even in their highest parts.

Many attempts have been made to account for the preference which tubercles exhibit for the upper parts of the lungs, but none of them satisfactorily explain it; they are based either on mere hypothesis, or the cause and the effect have been confounded. We confess our ignorance on this point, and can no more explain it than we can account for the preference shown by certain exanthematous and impetigenous affections for particular regions of the general integument.

Pulmonary tubercles pass through the different metamorphoses which are described in the First Volume.

1. They very frequently *soften*, and this change gives rise to *tuberculous suppuration of the lungs*, *tuberculous ulcer*, *tuberculous abscess*, (*vomica pulmonis tuberculosa*, *caverna tuberculosa*), and *tuberculous phthisis*. The separate, grey tuberculous granulations begin to soften in their centres, which become turbid, opaque, yellowish, and cheesy, and finally undergo purulent solution. The groups of tubercles break down at several points simultaneously, corresponding to the different separate tubercles of which they are composed. Hence, in the first case we have a small *primary tuberculous ulcer*; and in the second case, after the final solution of the whole mass, a much larger one, whose further progress we shall consider in the following remarks.

It is especially important to understand the manner in which the primary tuberculous ulcers *enlarge*, and give rise to such peculiar and extensive destruction of the lungs. This is elucidated by the process which goes on in the tissue surrounding

the softened tubercle. The breaking down of tubercles is always followed by a secondary deposition of tubercle in the surrounding parenchyma, the extent of this secondary deposition being proportional to the intensity of the general disease. Moreover these secondary tubercles and the tissue in which they are deposited likewise break down with a rapidity which stands in a direct ratio to the intensity of the dyscrasia, and in this way the tuberculous ulcer becomes enlarged. If this process goes on in so tumultuous a manner as to exhaust the powers of reaction and the supply of organic matter, the ulcer usually extends unequally in various directions, and forms an irregular, sinuous, and apparently lacerated cavern whose walls consist of lung-substance plugged up with softened tubercle, and whose internal surface presents, as it were, a gnawed appearance, without a trace of any inner lining except a coating of adherent, tuberculous pus. The small quantity of parenchyma occurring between the tubercles is in a state of compression and dirty brown discoloration (carnification), while that in the surrounding neighbourhood exhibits no trace of reaction, except a certain degree of hyperæmia. If this, as is usually the case, takes place simultaneously at several spots, two or more caverns will come in contact and will finally unite, and we then have either a number of caverns communicating with one another by means of sinuses of varying width, and either straight or tortuous in their course, or else the whole represents a large abscess with sinuosities in various directions. This cavity is intersected in various directions by bridges or rafters of rotten tuberculous lung-substance, which is likewise dependent in the form of shreds from its roof and walls.

This form of phthisis corresponds to the acute form of tuberculous intestinal ulceration, which runs its course without any reaction.

In other more common and less rapid cases, an inflammatory process, which must certainly be regarded as having a curative tendency, is established in the parenchyma around the softening tubercle or the primary tuberculous ulcer, and in the interstitial tissue amongst the secondary deposit. It gives rise to an albuminous greyish-white, or somewhat reddish, tenacious and viscid product, which occasions the closure and finally the atrophy of the air-cells, and is identical with

Laennec's *infiltration tuberculeuse gelatiniforme*. (See pp. 90, 91.) During this process the inner surface of the cavern becomes smoother and more uniform, and very often becomes covered with a thin greyish or greyish-yellow, thin, adhering investment of an apparently loose texture. This coating may sometimes, according to Andral, consist merely of the more solid portion of the pus contained in the cavity; in most instances this is, however, certainly not the case, but, as Laennec was the first to observe, there is a true exudation from the walls of the cavern like that which exposed or wounded animal tissues deposit on the surfaces of wounds or ulcers. This exudation is, doubtless, repeatedly thrown off, for if the process of tuberculous softening go on, neither it nor the adjacent wall of the cavern can meet with the conditions necessary for organisation; it melts or becomes disintegrated, and mixes with the pus in the cavern, and another membrane is formed in its place, so long as the tuberculous process on the one hand, and the reactive inflammatory process on the other, continue in the tissue in a certain antagonistic degree and proportion. The caverns enlarge, in the manner we have already described, by the softening and breaking down of the secondary tuberculous deposit in the tissue of their walls, and by the confluence of several neighbouring caverns into one. The parenchymatous bridges which traverse them are in a state of gelatinous infiltration, and contain tubercles, while externally they are coated by the above-named exudation.

In consequence of this process the lung-substance in the walls of the caverns becomes atrophied and converted into a more or less pigmentary, bluish-grey or blackish-blue, dense and tough layer of various thickness, the portion next to and lining the inner surface of the cavern being chiefly a whitish cellular tissue. To this the above described exudation adheres, and through both these shine the bluish atrophied parenchyma and the vessels which are laid bare and obliterated by the cavern, and which appear as yellowish-white ramifying streaks; scattered crude or yellowish softened tubercles may also be observed. These tubercles gradually soften, and lead, on the one hand, to a gradual enlargement of the cavern, while, on the other hand, they impede any comprehensive process of consolidation, since they perforate the cellular investment of

the cavity. The internal surface of the whole cavern is even and tolerably smooth, except at the spots where there are these new tuberculous excavations.

The pulmonary vomica in this condition is analogous to the tuberculous ulcer of the intestine with gelatino-lardaceous thickening of the submucous tissue on which it is situated.

The caverns naturally present the most manifold differences in reference to their size and number. The cases are not rare in which an abscess attains the size of a duck's egg, or of the fist, or even involves a whole lobe. When it is very large, the probability is that it has been formed by the confluence of several smaller caverns. The largest abscesses occur, with few exceptions, in the upper lobes, where, as we have already remarked, and especially in their upper third and at their apices, tuberculous deposits usually first occur, and where they first begin to soften.

It is a question of especial interest to ascertain how far the individual structures entering into the composition of the lung are involved in this destructive process, and above all, how the *bronchi* and *blood-vessels* within the tuberculous abscess are affected, what mode and form of destruction they undergo, and what destructive consequences follow when the cavern, in making its way outwards, finally reaches the *pulmonary pleura*.

The capillary bronchi undergo the same softening as the true lung-substance, for they, or at least their walls, are the seat of tuberculous deposition, and their mucous membrane becomes the seat of tuberculous infiltration (bronchial tuberculosis p. 34) during the softening of the pulmonary tubercle, just as we observe in the larger bronchial tubes in the neighbourhood of a tuberculous abscess. The capillary vessels become obliterated in the tubercle, and are exposed to the same softening process as the cellular strata surrounding them. If the cavern should now enlarge, the bronchial tubes become destroyed in the same proportion with the surrounding parenchyma, and it is only when the destruction of the tissue has attained a certain degree that an opening is effected into the tubes, and a communication established between the bronchi and the cavern. We only find bronchial tubes of a comparatively large size opening into the caverns, for the

smaller ones are compressed by the tubercle deposited in their walls and in their immediate vicinity, or by the products of interstitial inflammation, or they are closed by catarrhal tumefaction of their mucous membrane, or by tuberculous infiltration. Their mouths remain freely open in places where compression cannot affect them, in consequence of greater and more resistant thickness of their walls, or of their having a larger calibre. The number of bronchial tubes opening into a cavern is generally proportional to its size. They constantly open with a round or an oval fissure-like mouth, according as they are more or less transversely or obliquely situated in relation to the walls of the cavern, or are only ulcerated on one side. When the bronchial opening is recent, it commonly presents an ulcerated appearance, but subsequently, when the cavern has acquired a dense callous wall, it is bounded by a puckered border of mucous membrane in a state of gelatinous infiltration, which is analogous to the serrated, puckered, and similarly infiltrated border of mucous membrane which surrounds the callous tuberculous ulcer of the intestine. The mouth of the bronchial tube opens in exactly the plane of the wall of the cavern, and never projects beyond it.

The blood-vessels present, as it were, the very reverse condition. The bronchial vessels are usually obliterated and thrust aside, and run along the walls of the cavern as ligamentous, projecting, yellowish-white, ramifying cords, and those of an arterial nature, even when in this condition, for a long time resist the destructive processes which are here in operation. A partially or entirely obliterated vessel, enveloped in atrophied lung-substance, is usually found in the bridges which run across the cavity of the abscess. It often, however, happens that, before the vessels are obliterated, they are laterally denuded of the surrounding tissue and of their cellular sheath; the two inner coats then soon give way, and occasion the pulmonary hæmorrhages which, as is well known, occur in the course of phthisis.

There are two circumstances under which the caverns may reach the pulmonary pleura; either when they are originally formed in the peripheral portion of the lung, or when they were originally deep-seated, but have attained a considerable size in an outward direction. The first is very rarely dependent on the softening of tuberculous granulations, but, as we shall

presently show, is much more frequently a consequence of the softening and breaking down of tuberculous infiltration. In either case the pulmonary pleura may finally be destroyed, and this may occur in different ways and with different consequences. If there are no adhesions at the point where the cavern reaches the pleura, this membrane, after being denuded on its pulmonary surface, will be converted into a yellowish-white eschar, which extends over a greater or smaller portion of the cavern, and either becomes torn or else is loosened along its circumference and falls out in an entire piece; in this way tuberculous pus and atmospheric air find their way from the bronchial passages into the pleural cavity, and give rise to *pleurisy with pneumothorax*, and usually to speedy death. Mere cellular adhesions cannot prevent this termination; they are, in part, mechanically loosened by the effusion from the cavern, and being involved in the pleuritic process, they are, in part, likewise destroyed in the exudation. If, on the other hand, there are thick adhesions, that is to say if the lung is bound down by dense, thick, callous, cellulo-fibrous and fibrocartilaginous pseudomembranes, such as occur especially about the upper lobes and their apices in consequence of previous pleurisies, then the pulmonary pleura which has coalesced and become identified with these false membranes may sometimes be laid bare to a considerable extent without perforation and the above named consequences ensuing. But the tuberculous destruction is usually limited by these callous bands; cases occasionally occur in which even these are perforated; irritation is set up in them at one or more spots; they soften, become tuberculous, and suppurate, layer after layer; in this way they finally become perforated, and the tuberculous process attacks the wall of the thorax, insidiously advances to the ribs and soft parts, and at length reaches the outer surface of the chest, or even of the neck (Cruveilhier), in the form of a tuberculous sinus variously combined with caries of the ribs, the sternum, and the vertebræ.

Tuberculous Infiltration, when associated with the above-described metamorphosis of interstitial tubercle, usually softens with very great rapidity, and by hastening the progress of the disease, constitutes what is termed florid, or, by English writers, galloping consumption. It causes the most frightful destruction

of the pulmonary tissue, and gives rise to caverns of irregular form, which are surrounded by rotten, and as it were corroded parenchyma, infiltrated with tubercle and breaking down into pus. Tuberculous infiltration is most commonly deposited in the superficial portions of the lungs, and hence it is the caverns arising from this variety which most frequently open into the cavity of the pleura. There are several ways in which this may take place.

(a.) The pulmonary pleura may be puffed up by the air rushing into the cavern, and may be violently peeled off the tuberculously-infiltrated parenchyma for some distance beyond the extent of the cavern, so as to form a flattish, round bulla, which finally bursts.

(b.) It may be converted, as we have already shown, into a yellowish-white eschar, which either tears or becomes detached unbroken.

(c.) Both the pleura and the infiltrated parenchyma surrounding the cavern may be attacked with gangrene, and become changed into a dirty-brownish, or greenish, pulpy, shreddy, fetid mass.

This last-named termination is especially worthy of notice, as it may occur not only near the surface, but also in the deep-seated portions of the tuberculous infiltration, especially around a pre-existing cavern. Moreover, in consequence of the frequency with which intense tuberculosis of the bronchial glands is combined with tuberculous infiltration, it may occasionally happen that a communication may thus be established between a deep-seated pulmonary cavern and a cavern in a bronchial gland.

The *contents* of tuberculous caverns present many differences. Sometimes, and especially when the infiltrated tubercles begin to soften, these caverns contain a yellow and somewhat thickish pus; more frequently, however, they contain a thin, whey-like fluid (tuberculous ichor), in which may be observed numerous greyish and yellowish, friable, cheesy, purulent flocculi and particles, whose quantity, however, is not in itself sufficient to explain the profuse expectoration which so often occurs in phthisis. This fluid is often of a greyish-red, or reddish-brown, or chocolate colour, from the admixture of blood; or of an ash or blackish-grey colour, from the pigment

which it takes up during the softening of the tissue. Moreover, the caverns sometimes contain smaller or larger fragments of lung, resembling the parenchyma contained in their walls, and chalky concretions are occasionally found in them. In other cases they contain coagulated or fluid blood in various stages of discoloration.

This metamorphosis of pulmonary tubercle, in its twofold form, constitutes, as has been already observed, *tuberculous pulmonary phthisis*. If we now direct our inquiry to the state of the lung-substance around the tubercles and their abscesses, and from thence to the other organs and systems, in a distinct and uncomplicated case of this nature, we shall arrive at the following conclusions, in addition to what has been already stated, as the result of an anatomical examination when considered in reference to the living organism.

In the upper lobes, and especially in their upper third, there is usually a large cavern, surrounded inferiorly by several smaller ones, some of which communicate with it; between these are yellow tubercles in the act of softening, and grey tubercles just becoming opaque and discoloured, whilst in the lower portions, as well as in the inferior lobes, there is a comparatively small sprinkling of grey, crude, tuberculous granulations.

The lung-substance between the tubercles is found in various states, according to the progress made by the disease. It may be normal, but generally there is a vicarious emphysema developed in its superficial portions, while the deep-seated parts are not unfrequently hyperæmic, or in a state of œdema. It is, however, sometimes atrophied, and this is a more important change, owing in part to interstitial inflammation, in part to the obliteration of the bronchial tubes and air-cells, in consequence of the pressure exerted on them by the accumulated tubercles, and in part to the occlusion of the bronchi by the blennorrhœal mucous secretion when bronchial catarrh is simultaneously present. Inflammation (croupous pneumonia), which sometimes attacks the greatest portion of the non-tuberculous parenchyma, is also an important change; it appears partly as a brownish red, and partly as a greyish red hepatisation, which is everywhere converted into yellow tuberculous infiltration, which becomes dissolved, and collects in vomicæ; or the pneumonia may cause the deposition of a gelatino-

glutinous product. (See p. 84.) In well marked cases of this nature, the lung appears very bulky, and is coated with a greyish-yellow and generally thin pleuritic exudation, through which and the pulmonary pleura may be seen the peripheral tuberculous infiltrations, and the emphysematous patches amongst them.

In the *larynx* we find tuberculous ulcers, which vary in number and extent; and, associated with them, we find aphthous erosions, especially on the tracheal, and sometimes also on the pharyngeal mucous membrane.

The *bronchial tubes* proceeding to and from the caverns, exhibit streaks of mucous membrane in a condition of tuberculous infiltration, and are themselves filled with tuberculous matter; moreover, they are always in a state of catarrh, with reddening and softening of their mucous membrane, and with a muco-purulent secretion, which constitutes the greatest part of the sputa which are expectorated in the course of phthisis. The *bronchial glands* are enlarged, and more or less tuberculous.

Externally we find *pleurisies* which present great variety in their extent, and in the character, mode of organisation, and consequences of the exudation. They are the causes of the very acute pains in the chest to which phthisical patients are subject. Unless when they arise from superficial pneumonia, they are generally developed during the softening of the tubercles and the formation of the caverns, and are associated with the inflammatory reaction that is established in the adjacent interstitial pulmonary tissue. The most constant seat of these pleurisies is the conical apex of the pleura and the surface of the upper lobes generally; they thus correspond to the starting-point of tubercle and of its metamorphosis. They deposit an exudation which becomes organised into fibro-cellular cords, or into a thick, compact, fibrous investment, which covers the upper lobes from the apices downwards, in the form of a hood, and diminishes in thickness from above downwards, causing the lungs to adhere firmly to the costal walls, and thus affording a protection against the perforation that might otherwise be caused by large caverns.

With rare exceptions, we find *tuberculous intestinal phthisis* associated with pulmonary phthisis, and although the former is usually only a secondary affection, dependent upon the pul-

monary phthisis, it sometimes exceeds it in the rapidity of its progress, and rapidly occasions very great and exhausting ravages. As a general rule, the lower portion of the ileum is the part originally attacked, and from thence the ulcers extend upwards along this division of the intestine, and downwards over the colon; in their progress upwards the ulcers sometimes reach as far as the stomach. Frequently, however, and especially at certain times, the tuberculosis not only predominates in the colon, but is almost exclusively confined to it, the ileum entirely escaping; and sometimes we may readily perceive that the ulcers which are simultaneously present in the ileum are of a more recent date than those in the colon. In addition to the tuberculous ulcers on the intestinal mucous membrane, we also find that the corresponding *mesenteric glands* are more or less tuberculous.

The mucous membrane of the alimentary tract, especially of the stomach and large intestines, is also in a state of more or less developed blennorrhœa; and, towards the end of phthisis, an *acute softening* of the mucous membrane of the great *cul de sac* of the stomach is not of uncommon occurrence.

The *liver* is very frequently affected; the condition known as nutmeg liver, and depending on a morbid separation of the yellow and reddish brown substances, with a preponderance, and more or less fatty degeneration of the former, is extremely common, and so, also, is the true fatty liver. These changes in this organ are not peculiar to phthisis,—that is to say, to the softening of the tubercles and the tuberculous ulceration of the pulmonary tissue,—but are associated with tuberculous disease generally.

The *spleen* exhibits no constant change which stands in any essential connection with tuberculous ulceration of the lungs.

The right side of the *heart* appears sometimes to be dilated, in consequence of the impermeability of the lungs, induced by tubercle and its consecutive diseases; it is, however, much more frequently remarkably small, pale, and devoid of fat, in consequence of the anæmia which accompanies phthisis in its progress. In the former case we find stasis and accumulation of blood in the right side of the heart, and, from thence, in the whole venous system; in the latter, there is a general deficiency of blood, and a contracted aortic system.

The *central organs of the nervous system* exhibit no essential anomaly, although, as a consequence of acute phthisis, we not unfrequently observe hyperæmia of the brain and its membranes, and recent serous effusions into the ventricles, associated with white (hydrocephalic) softening of the cerebral substance.

The *muscles* are all emaciated in an extreme degree; the fat is, in most cases, almost entirely consumed, and the *cellular tissue*, especially on the extremities, is very often in an infiltrated condition.

Tuberculous pulmonary consumption is unquestionably *curable*, as we may infer from the appearances not unfrequently observed in the dead bodies of persons who formerly had more or less suspicious thoracic affections, and subsequently recovered. It is only by the investigation of the conditions under which these natural cures take place, that we can hope to arrive at a truly rational mode of treatment, and the results will be the more beneficial when directed against the tuberculosis generally, and not merely against the pulmonary abscesses. Pulmonary phthisis, or tuberculous ulceration of the lungs, can only be healed when the general disease, and consequently the local process on which the ulceration depends, is eradicated. There are incontrovertible facts to show that, under these conditions, pulmonary abscesses may actually heal in various ways.

a. The reactive inflammation of the interstitial tissue in the vicinity of the caverns gives rise, as has been already mentioned, to a gelatinous infiltration which causes an obliteration of the air-cells. By this means the whole of the adjacent parenchyma is converted into a dense, fibro-cellular layer of varying thickness. While this is taking place, the exudation, which is deposited by the same inflammatory process on the walls of the cavern, becomes organised from this fibro-cellular tissue into a smooth serous membrane. The whole cavern is now converted into a *cellulo-serous cavity*, whose inner surface secretes a serous, viscid fluid resembling synovia. The bronchial tubes, which open into these cavities, present a peculiar character, for the serous membrane lining the cavern, and the subjacent fibro-cellular tissue project beyond the outer stratum of the bronchial tubes at their openings, and their mucous coat hangs forward with a wrinkled, somewhat inverted free edge into the cavity.

More commonly, however, we find the caverns lined with a

villous, cellulo-vascular, more or less deep red layer resembling mucous membrane, which is intimately connected with the subjacent tissues. It appears in a constant state of irritation; and, as we generally find in caverns with which large bronchial tubes communicate, its conversion into a smooth serous membrane appears to be impeded by the irritation induced by the constant entrance of atmospheric air. An already formed serous investment may doubtless be again reduced to this cellulo-vascular mucous-membrane-like state, in consequence of this continuous influence. It secretes a turbid, muco-serous fluid, and it is not unfrequently observed to be covered with fresh exudations in consequence of higher degrees of irritation. It is extremely probable that these processes of irritation, associated with other causes presently to be described, effect the gradual diminution and finally the closure of the caverns. In cavities of this sort, the form of the bronchial openings is somewhat different from that which has been already described; for the bronchial mucous membrane coalesces with the lining texture of the cavern which is analogous to its own tissue, and they merge into one another without any apparent line of demarcation.

Ossaceous laminæ are sometimes developed under the serous investment, in like manner as in the cellular tissue beneath normal serous membranes.

In these caverns an event not unfrequently takes place, which very often proves fatal on its first occurrence; this is the *hæmorrhage* which is met with in caverns of this construction, and which always springs from the larger branches of the pulmonary artery traversing the walls; these branches often remain permeable, and become opened for a considerable extent on the side towards the cavity. There are two different conditions which may give rise to the opening of these arteries.

(a.) They either undergo an aneurismal dilatation in consequence of the absence of support in the direction towards the cavern, and finally tear at this point, without any further change in the texture of their membranes;—

(β.) Or the delicate cellular sheath of the vessel participates in the irritation of the adjacent investment of the cavern; the process extends to the fibrous coat, which becomes relaxed and infiltrated with gelatinous matter; and the vessel finally gives

way, a previous dilatation of its coats being sometimes but not always observed.

A circumstance deserving of notice sometimes accompanies these hæmorrhages. The extravasated blood in the cavern coagulates into a fibrinous clot, which completely fills it, and is attached to a pedicle, which is seated in the rent in the vessel, and is continuous in both directions with the cylindrical clot in the artery. The cavern may certainly contract around this clot of fibrin when, in the course of time, it has become shrivelled and finally cretified; but as the cavity in its previously described state must be regarded as innocuous, and may be closed in another and a simpler manner, this method of cure, except in cases where hæmorrhage takes place into a cavern which does not communicate with a bronchial tube, must always be regarded as dangerous, and only of actual use, insomuch as the fibrinous coagulum affords a support for the vessels in the walls, and prevents subsequent hæmorrhages, which might occur before the cavity had closed by the ordinary way.

The above described cavern must be regarded as a cured pulmonary abscess; but the cure may progress further, till there is perfect cicatrisation.

b. This occurs in the following manner:—If the abscess be not too large, it closes by a gradual approximation of its walls, which finally come in contact and coalesce. We then find, in place of the previous cavern, a cellulo-fibrous stripe, in which the bronchi end in blind sacs. This is of most frequent occurrence in the apices of the lungs, where the co-existence of open caverns and the presence of obsolete and cretified tubercles indicate the nature of the process that is here going on. The obliteration of a cavity of considerable size always occasions a corresponding depression of the surrounding parenchyma, and a cicatrix-like folding and puckering of the pulmonary pleura, which is most frequently and distinctly observed in the case of those cavities which are often superficially situated quite in the apices of the lungs. The thorax is also depressed to an extent corresponding with the size and number of the closing vomiceæ, obvious from the flattening and slight depression so frequently observed in the clavicular region.

(process is undoubtedly favoured very essentially by

certain circumstances, amongst which we may enumerate the local depression of the thorax, the contraction of its cavity in consequence of the diaphragm being abnormally pressed upwards by the contents of the abdomen, the development of emphysema in the parenchyma surrounding the cavern, and bronchial dilatation. It has been proposed and attempted to produce these conditions artificially, by way of treatment, in various and sometimes violent ways: we have already discussed (in the First Volume) the admissibility of these methods of treatment, their modes of action, and the consequences to which they may give rise.

When the healing process is rapid and continuous, the cicatrix sometimes encloses chalky concretions of various sizes, formed by the inspissation of tuberculous pus in the cavity.

(c.) The cavern, instead of cicatrising in the above described manner, may be filled up with a roundish or irregularly branched mass of fibrocartilaginous structure, in which the bronchi terminate in blind sacs. This is effected by the conversion of its cellulo-fibrous walls into a fibrocartilaginous callus, which continues to grow thicker. The cicatrix-like puckering of the surrounding parenchyma is generally in this case very inconsiderable.

This fibrocartilaginous mass may sooner or later be converted into a very compact osseous concretion of corresponding form and size.

2. The second metamorphosis which pulmonary tubercles undergo under favorable conditions, is their *cretification*. After their softening has began or is perfected, they gradually diminish in volume and become converted into a yellowish white, or greyish, or blackish grey, smeary chalky paste, and finally into a calcareous concretion. This concretion is situated, according to the intensity and extent of the process of reaction which is set up in the neighbourhood of the softened tubercle, either in obliterated pulmonary tissue, or in a fibro-cellular, or callous, fibrocartilaginous capsule. Here also cicatrix-like puckerings of the parenchyma occur over the cretified tuberculous masses.

Tuberculous infiltration may also undoubtedly undergo this metamorphosis, for we not unfrequently meet with paste-like masses of chalk, together with cretified tuberculous granula-

tions in the apices of the lungs, and corresponding in size and form to a pulmonary lobule; they are surrounded by a very delicate sero-cellular capsule, formed of condensed interlobular cellular tissue, and most probably are cretified *lobular*, tuberculous infiltrations.

3. Finally, pulmonary tubercle, when *in the form of crude grey granulations*, may become *obsolete*, shrivelled up, and abortive. It is then changed into opaque, bluish-grey nodules, having the resistant power of cartilage, which are incapable of any further metamorphosis. This destruction of the tubercle is either general, or it is combined with the process of cretification, the central portion or nucleus being converted into a chalky concretion encysted in the obsolete peripheral layer of tuberculous matter.

From what has been already stated, it follows that pulmonary tuberculosis may be cured by phthisis with the elimination of the tubercle; but the two last described metamorphoses, opposite as they are to one another, constitute more direct healing processes. Any one of them may take place under favorable conditions, and, as a general rule, they are all found in one and the same individual, for we find associated together cellulo-fibrous caverns, their cicatrices, and cretified and obsolete tubercle. They are generally all found imbedded together in obsolete parenchyma, infiltrated with black pigment.

Tuberculosis is either an *acute* or a *chronic* disease. In *acute* cases it attacks both lungs simultaneously, and frequently other parenchymatous organs and membranes, giving rise to peculiar symptoms resembling those of typhus; the tubercle is the product of tuberculous dyscrasia of the blood developed in a very high degree. The tuberculous mass is, in some cases, deposited at once, and in others at different intervals, which rapidly succeed one another, and are indicated by paroxysmal exacerbations; it is formed of grey, crude granulations, which are either very minute, vesicular, and transparent, or, in some cases, as large as millet-seeds. The tubercles are always very numerous, discrete, and uniformly scattered through the lung-substance; it is only rarely that we find them accumulated and confluent at individual spots, and in these cases they are all in the same stage, namely, that of crudity. Moreover the lung is in a state of hyperæmia, œdema, and emphysematous

textural relaxation; the hyperæmia occasionally passes into pneumonia and hepatisation.

In most cases it only attacks the lungs after tuberculous disease has advanced in them to the stages of softening and ulceration (*vomica*), and after it has existed for a longer or shorter period in its favorite locality—the apices of the lungs—in the state of more or less circumscribed, insidious tuberculosis. A pre-existing chronic tuberculosis of the lungs is generally the predisposing cause of the acute production of tubercles in those organs. It proves fatal in consequence of the hyperæmia and of the subsequent œdema to which it gives rise, in consequence of the violent production of emphysema, or from paralysis of the lungs.

Chronic tuberculosis either deposits its product imperceptibly, or else as crises of a mild general disease, with symptoms of moderate vascular excitement, and recurring at intervals. In accordance with this view we find tubercles of various ages and stages; and at the extreme points of the diseased lung-substance we have the two extreme stages of tubercle; at the apices, where the tubercles are first developed, we have caverns; and at the lowest portion we have recent, crude, tuberculous granulations; between these we have dissolved tubercles next to the caverns, and lower down such as are just beginning to soften.

It either proves fatal in the form of phthisis through exhaustion and tabes, or through some of those accidents which we have already described as liable to occur in the course of phthisis, as for instance the supervention of pneumonia with a tendency to tuberculous infiltration (hepatisation), hyperæmia or œdema of the lungs, hyperæmia of the brain and serous effusion into its ventricles (hydrocephalus, serous apoplexy,) tuberculous meningitis, exudative processes in the neighbouring mucous canals, as the trachea or œsophagus, purulent metastases, or the supervention of acute pulmonary or general tuberculosis.

The tuberculous habitus in general, and more especially the irritable scrofulous habitus, are the stamp indicating a predisposition to pulmonary tuberculosis; the torpid scrofulous habitus more commonly gives rise to bronchial tuberculosis. The well-known (phthisical) conformation of the chest which predisposes to pulmonary tubercles, is by no means invariably

present; its peculiar relation to tuberculosis is unknown, and any connection between the smallness of the respiratory organs in a contracted thorax, and the development of pulmonary consumption, is only hypothetical. Tubercles are often developed in the lungs of individuals, independently of any marked external influences, and then form *constitutional pulmonary tuberculosis* and *pulmonary phthisis*. On the other hand, they may arise, independently of this constitutional dyscrasia, in consequence of appreciable noxious influences, which induce either a purely tuberculous condition of the juices, or a modification, that is to say, a combination of this state with some other. This is *acquired tuberculosis*, which is either *pure*, or more or less *modified and combined*, such as follows the exanthemata and impetigo, gonorrhœa, syphilis, and anomalous gout, and occurs in drunkards, after the suppression of normal or habitual discharges, as, for instance, of the menses, after the cure of inveterate ulcers, &c.

The inveterate forms of dyscrasia deposit different varieties of tubercle, which have not hitherto been fully described; they occasionally terminate in *hæmorrhagic tubercle*. The tuberculosis is distinguished from the ordinary forms by commencing at an uncommon part, by its unequally attacking the most different parts of the lungs, by the deposit being accumulated in circumscribed or grape-like branches, by its very considerable amount, and by its peculiar, dirty-grey or leaden colour with a greenish sparkling appearance. (See Vol. I.)

Cancer of the lungs is sometimes deposited in a form resembling tubercle; we must carefully avoid confounding these morbid growths.

Pulmonary tuberculosis, like tuberculosis in general, is excluded by all the conditions enumerated in the First Volume, especially by diseases of the lungs, attended with atrophy, emphysema, bronchial dilatation, excessive condensation, compression, obsolescence or obliteration of the tissue.

6. *Cancer of the Lungs*.—Cancer occurs in the lungs both in the form of *carcinoma medullare* and *carcinoma fasciculatum, seu hyalinum*. The latter is extremely rare, but the former is comparatively common, and it is to it that the following observations apply.

a. It most commonly occurs in the form of *roundish*,

separate masses, varying from the size of a hemp-seed to that of the fist, and occasionally being even larger, and enclosed in a very delicate cellular capsule; they are composed of gelatinolardaceous or lardaceo-encephaloid or true encephaloid parenchyma, and hence they vary considerably in consistence; they are usually white, but are sometimes of a greyish red, or dirty yellowish grey colour. They are generally scattered in very considerable numbers throughout the lungs, both near the surface and deep in the texture, and when they are contiguous to the pulmonary pleura, they undergo a flattening or depression. The injury of the surrounding parenchyma is limited to its being displaced and compressed in the immediate vicinity of the adventitious product. It is only very seldom that it undergoes ichorous disorganisation, in which case the accumulated cancerous ichor makes its escape by communicating with the bronchi. It usually proves fatal by the exhaustion induced by its excessive growth, and by the high degree of general cancerous cachexia from which the growth originates. Pulmonary œdema and hydrothorax commonly supervene, either with or without simultaneous cancer of the pleura.

It very rarely occurs in the lungs as primary cancer, that is to say, as the first in a series of successive local cancers; it almost always exists in association with other, and generally many cancerous deposits of older date, distributed over several organs; and is often developed with great rapidity after the extirpation of large cancers. It is chiefly combined with cancer of the pleura, with which it is usually simultaneously developed, or with cancer of the mediastinum, or of the mammary gland, the liver, the kidneys, or the osseous system.

b. The occurrence of pulmonary cancer as a *special form of tubercle* is very rare, and is never met with unless when there is cancer in some other organ. It presents itself in the form of tubercles or nodules of the size of a millet or hemp-seed, which, as far as we yet know, may be distinguished from true tubercles by their bluish white colour, their softer consistence, their aggregation in groups, and a difference in their elementary structure and composition. They sometimes exist in association with a *retrograde genuine pulmonary tuberculosis*.

c. Cancerous matter is very rarely *infiltrated or effused into the air-cells*. When it occurs in this form it is the product

of a pneumonic process, which, under the influence of a dyscrasia excited by the extirpation of cancer, assumes the external characters, and the elementary structure of carcinoma; the lung in this case appears hepatised with cancerous matter.

Medullary cancer of the lungs is sometimes more or less blackened by a pigment which enters into its composition; the medullary nodules are marked with brown, blackish-blue, violet, or black spots or stripes, or are completely and thoroughly black, constituting melanotic cancer—cancer melanodes—of the lungs. We have never met with it except in association with general and, in fact, with very acute medullary cancer.

SUPPLEMENT.

1. *Diseases of the Thyroid Gland.*—As a general rule, the thyroid gland is liable to few diseases, and of these diseases we are almost as ignorant as we are regarding the structure and the function of this organ.

It very frequently presents anomalies of *size*, being often very much enlarged. The *augmentation of size* is sometimes transitory and rapid, as, for instance, when it depends upon congestion or inflammation, and sometimes in the case of lymphatic goitre; or there may be a persistent gradual increase, as is observed in the more advanced stages of goitre. It either attacks the whole gland uniformly, which then retains its original shape, or one lobe only, or a small part of one may be the only portion affected, so that the pressure which the gland naturally exerts on the trachea and larynx is variously increased in extent, and may affect not only the pharynx and œsophagus, but also the great vascular and nervous trunks on both sides of the neck, and even the trachea and bronchi, and the blood-vessels within the thorax. Those forms of enlargement are rarer, but at the same time more important, in which the thyroid gland tends to surround the œsophagus like a ring, and in which the isthmus grows downwards so as to form a middle lobe, which descends along the trachea behind the manubrium sterni into the thoracic cavity; in the latter case it becomes transversely contracted when opposite

the semilunar notch, but expands immediately below it, (*asthma thyroideum*.)

The *diminution of size* or *atrophy* of the thyroid gland is an affection of little interest.

Hyperæmia of the thyroid gland is not unfrequently observed, and most commonly occurs when there is some mechanical impediment to the emptying of the vena cava descendens and of the right side of the heart. Under these circumstances it may be either transitory or persistent. It may be recognised by the dark colour of the gland, its abundance of blood, its looseness of texture, and its swollen condition, (*hyperæmia, congestive turgescence*.) *Apoplexy* of this gland, when its texture is normal, is extremely rare.

Inflammation of the thyroid gland, as a *primary* affection, is of very rare occurrence, at least as an object of anatomical observation. But we sometimes find what are termed metastatic *abscesses* in it, especially when there are numerous similar deposits in other organs, consequent on puerperal uterine phlebitis. Abscesses of the thyroid gland may give rise to a deposition of pus in the mediastina, or they may open into the trachea, or, which is most commonly the case, they may enter into the œsophagus on its left side.

The most common disease of the thyroid body is that to which we apply the word *struma* (using the term in its strict signification), and its most striking characteristic is, as we have already mentioned, an augmentation of size. In the slighter degrees in which it usually occurs, it presents a very simple change of texture depending on a more decided development of the cellular structure of the organ. This occurs either equally through the whole gland, which then everywhere contains cells of equal size, or else we observe one, several, or very many isolated or agglomerated cells larger than the others, which are converted into roundish elongated cysts, with delicate membranous walls, and contain a gummy or glue-like, yellow, brownish or greenish matter (*colloid*). If this matter has attained a certain consistence, the cut surface of the gland presents a lardaceous appearance, and communicates a peculiar waxy and doughy feeling; the organ is at the same time pale and anæmic, and presents a marked increase of size without any disproportion of form.

There are certain unknown conditions under which, on the one hand, the secretion contained in the dilated cells undergoes modification either from the beginning or during the progress of the disease, or, on the other, the walls undergo a striking change. In the former case we find gelatinous or albuminous substances, of a whitish, grey, or flesh-red colour deposited in the form of concretions, whose coat may be peeled off, or they fill the interstices of an extremely delicate cellular network of new formation. In the latter case the walls of the cells increase in thickness, and the cells become developed (hypertrophied) into sero-fibrous cysts, which may contain various matters besides those already named, and which often attain an astonishing size. These changes constitute those forms of struma, which are known as *struma lymphatica* and *struma cystica*.

There can be hardly any doubt that these processes are essentially based on irritation, for repeated inflammations attack the walls of the dilated cells, and especially of the above named cysts, during the ordinary progress of the disease, although they doubtless often pass unnoticed. Here, as on normal serous, and fibro-serous membranes, they deposit the most varied exudations, and in consequence of the newness of the tissues, these are often hæmorrhagic, and accompanied by the separation of large clots of fibrin. These, together with the walls of the cyst, undergo all the same metamorphoses as occur in the exudations and the walls of normal serous sacs, (See Vol. III,) even to chalky transformation and ossification. The cysts in this manner not unfrequently become perfectly obliterated by contracting around the exudation, and we then find tough, somewhat voluminous, nodular, osseo-cartilaginous, chalky concretions imbedded in the gland.

True effusion of blood not unfrequently takes place into the cavities of the dilated cells and of the cysts.

The tendency to *cyst formation*, exhibited by the parenchyma of the thyroid gland, extends in a remarkable manner to the adjacent cellular tissue, for in no situation do we so frequently meet with small or large cysts with serous, gelatinous, or glue-like contents, as in the neighbourhood of this organ.

All other adventitious growths, excepting the above named serous, fibrous, cartilaginous, and bony productions, are ex-

tremely rare in the thyroid gland; thus *tubercles* are scarcely ever found in it, and *medullary cancer* only very rarely.

2. *Diseases of the Thymus Gland.*—Anomalies of the thymus gland are even rarer than those of the thyroid body; the only abnormal conditions with which we are at present acquainted are a more or less considerable increase of its size in new-born children, and its persistence to the fifth, sixth, or seventh year, or even to or beyond the age of puberty. Its abnormal enlargement is almost entirely restricted to children in whom we simultaneously observe a great predominance of the whole lymphatic glandular system, rachitis, and hypertrophy of the brain. It presents either two lateral, flattish, round, thick lobes, which descend on each side into the mediastinum posticum, or it forms a tongue-shaped mass which extends downwards on the pericardium, and rests on the right auricle. Whether the *thymic asthma* which has been recently described, and which occurs in delicate children, is actually dependent on the pressure of an enlarged thymus on the air-passages, or whether there is any essential connection between that disease and the thymus, are questions requiring additional observations and careful examination.

PART II.

DISEASES OF THE ORGANS OF CIRCULATION.

PART II.

ABNORMAL CONDITIONS OF THE ORGANS OF CIRCULATION.

WE may divide the above into Diseases of the Heart, including those of the Pericardium, and Diseases of the Arteries, the Veins, and the Lymphatics. Under the last head are included Diseases of the Lymphatic Glands.

I.—ABNORMAL CONDITIONS OF THE PERICARDIUM.

§ 1. *Deficiency and Excess of Formation.*

The *first named* species of malformation manifests itself as a deficiency of the pericardium, occurring generally when the heart lies outside the thorax, although it is also met with when this anomaly is not present; but is then of less frequent occurrence. This deficiency is in almost every instance merely partial, consisting in the congenital anomalous position of the heart outside the thorax in a fissure of the pericardium, although it is not uncommon in some cases to meet with less marked traces of the same condition in the region of the larger arterial trunks and along the right layer of the mediastinum. The heart and the left lung lie, as a general rule, in one common large serous sac, which gives rise, at the place from whence the arterial trunks emanate, to the above mentioned rudiments or traces, in the form of fatty mesentery-like folds.

The apparent deficiency of structure, induced by the firm adhesion of the pericardium to the heart, seems to have been mistaken by some older observers, for true deficiency of structure.

An Excess of Formation occurs in double monsters, where the pericardium is found to contain a double heart.

PART II.
DISEASES OF THE ORGANS OF CIRCULATION.

been generally described. Their free surface commonly appears as if covered with villous threads, which are either soft and lax, or stiff, and vary in character. Laennec has compared them to the inequalities remaining on two plates, which, after having been covered with a layer of butter, and laid against one another, have been quickly separated, and it is probable that this appearance gave rise to the terms made use of by the ancients, when they described the heart as *cor villosum*, *tomentosum*, *hirsutum*, *hispidum*, &c. Sometimes these shaggy masses are more or less accumulated at different spots, or ranged side by side, which is doubtlessly owing to the direction of the undulations produced in the serous effusion by the heart's motion. In many cases they may be aptly compared to the appearance presented by the dorsal surface of a bullock's tongue, whilst in others, the coagulum exhibits an areolar free surface, similar to that of the mucous membrane of the gall-bladder.

When the coagulable matter occurs in *larger quantities* in the serous portion of the effusion, it is found in some few cases in the form of roundish and somewhat flattened free bodies, about the size of a bean or hazel-nut, and generally constituting a network between the heart and pericardium, to both of which it adheres.

The plastic coagula become converted into a cellular or cellulo-fibrous dense tissue, with a permanent thickening of the pericardium, corresponding to the intensity of the process; and the different loose filamentous adhesions, or close fusions of the heart and pericardium, which are so frequently observed, either partially or totally (according to the extension of the process) are formed by this tissue. Amongst the partial adhesions, we may specially draw attention to those of a circumscribed nature occurring at the apex of the heart, those occurring at different parts along the sulcus transversalis, and the adhesions of the pericardium in the vicinity of the arterial trunks. In the first of these spots, the connecting medium is often drawn into long threads or strings by the movement of the heart's apex, and the adhesion is thus at length broken through, in consequence of which we usually find an accumulation of long, shaggy, cellular tissue at that part, and on the opposite portion of the pericardium; the second class of

adhesions derive importance from their ordinary combination with diseases of the valves, especially towards their margin of insertion; and the last from the evidence which their common occurrence affords of the frequency of pericarditis, which may prove of serious moment at the origin of the large vessels, as we shall subsequently have occasion to consider.

The *milk-spots*, or *maculæ albidæ*, are appearances of frequent occurrence on the heart. They are occasionally met with on the inner surface of the pericardium, but most frequently on the serous investment of the heart. There can scarcely be a question but that they are products of a partial or circumscribed process of inflammation. They are pale, bluish-white, tendonous-looking spots or *plaques*, appearing, when closely investigated, to be glued or soldered to the subjacent tissue; on being torn or detached, the pericardium is brought into view, and is almost normal in its character,—not perfectly smooth, but having a dense and sometimes even an opaque tissue. They must be distinguished from many other diffuse opacities of common occurrence on the pericardium, which consist in an inconsiderable excess of structure,—hypertrophy, a slight thickening and condensation of the serous investment of the heart. They further manifest many different characteristics with respect to their size and distribution, the number in which they occur, their form and limitation, their surface and mode of attachment; finally, they occur more frequently on some portions of the heart's surface than on others.

The *size* of these spots varies from that of a silver groschen, or a narrow stripe, to that of a silver thaler or more, so that one spot often spreads over a great portion of the heart's surface. A number of these spots are often found together, and they then blend into one another. Their *form* varies very much, and is extremely irregular; they commonly occur as narrow stripes along the coronary vessels in the sulcus longitudinalis. They usually expand into linear projections at their periphery, and are either sharply defined, or gradually attenuated into a very delicate membrane, as may be seen in moist preparations. Their *surface* is either smooth, even, serous and shining, or wrinkled, folded, pale, felt-like, and shaggy; the whole presenting a layer of newly formed cellular tissue.

These milk-spots occur on every portion of the heart, but they are certainly more frequent on the right than on the left ventricle; they usually appear on the auricles in the form of stripes, and finally are met with at the origins of the arterial trunks, and more especially at that of the aorta.

In connection with the subject of milk-spots, we may notice the metamorphosis of a partial exudation into *fibroid granulations*, occurring about the size of a millet-seed. These are especially to be met with on the auricles, and on the corresponding portion of the parietal surface of the pericardium. Granulations of this nature are frequently situated on these spots.

Inflammations with purulent exudation are distinguished by the quantity of the effusion, and are important on many accounts, which will be subsequently considered.

Scattered accumulations of pus are of very frequently occurrence in the sub-serous layers of the pericardium.

It is only in very rare cases that the purulent exudation leads to suppuration of the pericardium.

Our museum presents an instance of an originally sero-purulent exudation, which appears to have gradually undergone the following remarkable metamorphosis. At the circumference of the left side of the heart, the pericardium is closely adherent, whilst a whitish, very turbid fluid resembling milk of lime is accumulated around the right side of the heart. The inner surface of the pericardium, and more particularly the outer investment of the heart, appear partially encrusted as with a sandy mortar, and partly covered with a white, smooth, gypsum-like coating.

Among the *chronic inflammations* those attacking the pseudo-membranes are especially frequent and important; here we find peripheral coagula of very considerable thickness, density, and power of resistance, and of a fibroid texture; the pericardium itself acquires a considerable degree of thickness, and in cases where there is a resorption of the fluid, the two lamellæ of the peripheral coagula adhere together, and the heart becomes enclosed in a thick, tough, unyielding casing. *Hæmorrhage* very generally accompanies the secondary exudations in this process.

Finally, when the necessary conditions are present, the exuda-

tion may be *tuberculous*; but this subject will be noticed when we proceed to the consideration of tuberculosis of the pericardium.

In every form of pericarditis the pericardium may become very much distended, in consequence of the great quantity of the exudation, and especially of its serous portion.

Osseous concretions are not unfrequently developed in the dense fibroid exudations occasioned by the process of chronic inflammation recurring in the pseudo-membranes. We shall have occasion to return to this subject in a future page.

2. In reference to the *secondary effects* produced in the organism by general pericarditis, among which we must especially place extensive inflammations of the large serous sacs, it is worthy of notice that several, as for instance cachexia and dropsy, usually occur at an early stage and in a high degree of development. These conditions are occasioned by the injurious influence exerted by the pericarditic process on the heart, in consequence of which the muscular substance of that organ is paralysed, its colour changed to a dirty brown or yellow, and a flabby condition induced, which admits of the texture being easily torn, and which speedily leads to (passive) dilatation of the heart. These phenomena are collectively the more striking in proportion as the pericarditis is chronic, and the exudation is purulent, hæmorrhagic, or tuberculous; the dilatation becomes more permanent, the more completely the coagula have been metamorphosed into a thick dense resisting tissue surrounding the heart.

Pericarditis, more especially when of a chronic form, is important in reference to the origins of the large vessels. It would seem, according to our view, that this disease, as far as it affects the cellular sheath of the vessels in the sub-serous cellular tissue, must induce paralysis of the elastic coat, dilatation of the aorta, and that form of spontaneous laceration of the vessels within the pericardium, which is so often found to occur.

3. Pericarditis frequently occurs in original combination with inflammation of other serous sacs, as for instance with pleuritis, inflammation of the synovial membranes of the large joints (Bouillaud), and very frequently with pneumonia. It is, moreover, in like manner associated with endocarditis, and

occasionally with carditis: during the later stages it is often accompanied with the first named inflammatory processes, and also with meningitis.

Pericarditis is often a secondary affection associated in various degrees of intensity with other processes of exudation, and very frequently a slight reddening, injection, and an inconsiderable degree of effusion, are found to attack the pericardium at periods of complete exhaustion, and in consequence of extensive exudations. Acute pleurisies extend from the mediastinum to the pericardium, and centres of inflammation in the muscular tissue of the heart sometimes occasion general and sometimes partial inflammation of the pericardium.

Pericarditis, contrary to the results of the investigations of many observers, is frequently met with beyond the middle periods of life and even in advanced age.

b. Secondary Formations.

1. *Adiposity of the Pericardium.*—We not unfrequently observe an excessive accumulation of fat on the pericardium. This occurs in general not only in conjunction with an excess of fat in the heart itself, but also together with fatty accumulations in the abdomen, that is to say in the great omentum and its appendages, in the mesentery, under the costal pleura, &c., constituting a general condition of corpulence.

2. *Fibroid Tissue* occurs in the milk-spots and in the fibroid granulations, assuming the form of a thick exudation, having the property of resistance, and being of a very dense texture.

3. *Anomalous Osseous Substance* is scarcely ever developed, excepting in the above named fibroid exudation, after the lamellæ have become fused together, and the pericardium has thus been made to adhere to the heart by this dense and resisting fibroid medium. This adhesive stratum is now covered by a deposit of tuberos uneven laminæ and bands, or thick, roundish, nodular masses. The space occupied by the deposits varies considerably, but the first named of these forms of deposition occasionally extends so far as to cover the greater portion of one ventricle. The projections occupying the side next the heart frequently extend to the texture of the heart itself, displacing the muscular bundles, and appearing as if developed within them. The thick, round, nodular masses

are generally observed in the neighbourhood of the sulcus transversalis on the left side of the heart, being connected with an osseous concretion which usually has its seat at the margin of insertion of the mitral valve. They are consequences of a former state of endocarditis combined with pericarditis.

4. *Tuberculosis of the Pericardium.*—Tuberculosis rarely manifests itself in the pericardium in any other form than as a product of inflammation. Pericarditis gives rise to an exudation, whose peripheral coagula, after passing, wholly or in part, through various metamorphoses, merge into tubercle. It frequently happens in chronic inflammations of the exudation-deposits that the deeper or older strata have become tuberculous, while the more recent coagulum, which is becoming tuberculous, is covered by a secondary, villous, and shaggy deposit from the fluid effusion.

This form of tuberculosis of the pericardium, in accordance with what has been already stated regarding tuberculosis of the serous membranes generally, is not of a primary character, being usually associated with and dependent upon an earlier tuberculous condition, which has formed as it were the focus or starting-point of the disease, and has been manifested as tuberculosis of the lungs and bronchial glands, or as chronic tuberculosis of some of the great serous membranes, especially of the peritoneum.

In this form of pericarditis there is always much serous effusion, which is undoubtedly increased by the inflammation being paroxysmally developed in the tuberculising coagulum. This effusion frequently becomes hæmorrhagic, in consequence of such secondary exudations.

The tubercles, which are often of considerable size, and fused together into one aggregate mass, are occasionally seated close to the muscular tissue, into whose fibres they occasionally penetrate so far as to lead to much doubt regarding their original position.

This form of tubercle very rarely passes into the metamorphosis of complete softening, since death, when it ensues, is generally occasioned by the pericarditis, or the subsequent tuberculous secretions, or even by general cachexia. Occasionally one or more tubercles, or tuberculous masses, may certainly be observed to become disintegrated, but the process

is seldom sufficiently prolonged to produce an abscess, or a corrosion,—tuberculous suppuration of the pseudomembrane and of the pericardium itself. As we have already observed, the tuberculous exudation soon manifests its influence on the tissue of the heart, which, however rapidly the disease may prove fatal, is always found to be strikingly discoloured, having generally acquired a dirty-brown colour, and is moreover flabby and easily torn.

5. *Cancer* only affects the pericardium in a secondary manner; and, in most cases, only where secondary cancerous formations have been developed in the mediastinum. This secondary mass either spreads itself in the form of an infiltration of the fibrous layer of the pericardium over a large portion of its surface, or presses upon and into the tissue itself, where it becomes developed into roundish or flattened, teat-like nodules.

In the very rare cases in which cancer occurs in the pericardium, independently of the above conditions, it presents itself in the form of numerous, flattened, and roundish nodules. It then always occurs in combination with cancer of other serous membranes, especially of the contiguous pleuræ, and depends upon an excessive dyscrasia, developed by previous cancerous degeneration of different parenchymatous structures, and frequently exasperated by the eradication of large carcinomatous masses.

We have never met with any other form of cancer of the pericardium but the medullary.

§ 5. *Anomalies of the Contents of the Pericardium.*

Besides the anomalies already treated of, it remains for us to notice, among those which exhibit special points of interest:

Blood in a fluid or coagulated condition. It is almost always an arterial extravasation, and has been deposited by the spontaneous rupture of the left ventricle, or by a laceration of the origin of the aorta, occasionally also as the termination of an aneurism. The quantity extravasated seldom amounts, under these circumstances, to more than from 2 to $2\frac{1}{2}$ lbs.

Serum is frequently accumulated in greater excess than the normal quantity, which varies from $\frac{1}{2}$ oz. to 1 oz.; and it then constitutes *Hydrops Pericardii*. This accumulation becomes serious in proportion to its amount, and possesses greater im-

portance where the other co-existing anomalies, by which dropsy is influenced, are inconsiderable. It is generally combined with dropsy of other serous sacs, anasarca, and œdema of the lungs, and usually has a common origin with them. Occasionally it predominates over these affections, as is especially the case in pulmonary phthisis. The period of its duration may be estimated by the extent to which the fat has disappeared from the heart, and its place been occupied by a serous infiltration of the cellular tissue, and in proportion to the extent of turbidity and swelling from imbibition, observed in the pericardium, and especially its outer surface, and to the decoloration and paleness of the substance of the heart.

The quantity of the *Liquor Pericardii* is often strikingly small, amounting to no more than what is barely sufficient to moisten the pericardium and the heart itself. The pericardium may even in some parts appear perfectly dry, of a yellowish colour, and resembling parchment. We have remarked this appearance, which is devoid of importance, on the lateral portions of the pericardium, principally on its left side, where it had been brought in contact with the anterior parts of a lung in which emphysema had been developed.

We have never met with an accumulation of *Air* in the pericardium, *Pneumatoxis Pericardii*. Most of the cases recorded, like the pneumatoses of other serous sacs, leave room for many doubts regarding their existence during life.

Free Bodies are of very rare occurrence in the pericardium. In a very remarkable case of pericarditis we discovered, in the serous effusion, numerous fibrinous, soft, yellow concretions of the size of beans or almonds, and similar to the latter in shape, which would no doubt have eventually been converted into elastic, tough bodies of fibroid tissue.

II.—ANOMALIES AND DISEASES OF THE HEART.

We will now proceed to consider the Anomalies and Diseases of the Heart, including those of the Valves; but wherever it may prove of great practical interest to acquire a more correct knowledge, both generally and specially, of the anomalies of the valves treated of in the different sections, we purpose, at the close of each, entering more fully into the details of the subject.

We will, moreover, consider simultaneously all original malformations of the heart and of the vascular trunks, not only on account of the natural connection existing between them, but also with a view of furnishing the premises necessary for the better comprehension of the appendix on cyanosis, which is subjoined to our remarks on the anomalies of the heart. In order, as far as possible, to facilitate a reference to the most important original malformations, we have arranged the following sections somewhat differently from those by which they are preceded.

§ 1. *Deficiency and Excess of Formation.*

Absence of the Heart—Acardia—is generally of very rare occurrence, but is a common phenomenon in *Acephalia* (absence of the brain), especially where there is an absence of the upper half of the trunk. It has only been observed in very rare cases when the nervous system is perfect and complete.

In the consideration of deficiency of the heart we include a series of deficient formations (arrested developments) which may be arranged as follows.

a. The lowest type of formation is that in which a single cavity without valves represents a ventricle in which a dilatation of the vena cava appears as the rudiment of an auricle. The latter is membranous, and the former has only thin muscular walls and weak trabeculæ.

b. Next we have a heart consisting of one ventricle and one auricle, with simple vascular trunks, into the former of which opens an aorta, and into the latter a vena cava. In many cases this formation approximates to the succeeding one in which there are two auricles with a single ventricle.

c. In this form there is a single ventricle and one auricle which is either partially or wholly divided into two cavities by means of a partition wall. The arterial and venous trunks may be either single or separated.

d. Here a capacious ventricle presents the rudiment of a septum ventriculorum, which becomes so far developed as finally to exhibit only an aperture which is usually situated at its upper extremity. The most common anomaly of the vascular system combined with this form is the origin of the aorta from both ventricles, and the displacement of the pul-

monary artery. The foramen ovale in the partition between the auricles remains open. In other cases the septum is perfect, but so situated as considerably to diminish the size of one or other of the ventricles, interfering with its valvular apparatus, and giving the auriculo-ventricular opening a very contracted and even closed appearance,—a condition of things that involves the patency of the foramen ovale and of the ductus arteriosus.

e. Here we have a form of the heart in which the partition between the auricles is defective, although there is a perfect separation of the ventricles. The degree and form of this defective structure are very variable. The septum is sometimes entirely absent, its line of direction being simply indicated by several soft membranous filaments which pass from the posterior to the anterior wall of the common cavity of the auricles. In other cases the rudiment of a septum atriorum develops itself in the form of a crescentic band, either from the arch of the auricle, or below from the septum ventriculorum. The wide aperture of communication between the two auricles is round or oval, and has its major axis inclined from before backwards. In other cases, the rudimentary structure is sometimes so far developed round the septum that this deficiency is often represented by a smaller and obtuse triangular aperture; and in other cases, again, the septum seems so far developed from above that it may easily contain a foramen ovale. There are, in this case, two apertures in the partition between the auricles, the former, which depends on defective formation, is not closed, and the latter (the foramen ovale) remains open.

Cases of this nature are generally characterised by a congenital contraction or insufficiency of the aorta, by extraordinary dilatation of the pulmonary artery, and by eccentric hypertrophy of the right side of the heart.

f. In this form the foetal passages,—the foramen ovale, and the ductus arteriosus,—remain open. The degree of the patency of the foramen ovale varies considerably, its valve being very nearly or entirely absent in some cases, but more commonly the upper third or fourth portion of it is wanting, and most frequently of all there is a mere deficiency of attachment at the upper part of the isthmus, by which means a fissure rather than a foramen is formed, which communicates in a very oblique direction from below and behind upwards and

forwards from the right into the left auricle. This foetal condition is sometimes persistent to a greater or less degree, and consists in this—that under a marginal projecting rudiment of the Eustachian valve, which penetrates into the anterior columna isthmi fossæ ovalis, there is a communication with this fissure-like aperture, or with the still patent foramen ovale. The opening at the upper boundary of the isthmus is either formed as a simple fissure, or consists of several small and roundish apertures. The cause of the patency of the foramen ovale frequently depends on the different malformations of the heart already enumerated, and on the different anomalies of the arterial vascular trunks and of the ductus arteriosus, which still remain to be noticed. The patency of the foramen ovale most frequently corresponds with an incidental arrest of development. In some cases it is associated with smallness of the heart, and retraction of the apex (the foetal condition). It is of very frequent occurrence in its lesser degrees.

The patency of the ductus arteriosus will be more fully noticed in a future page.

The following are the most important of these *Anomalies of the Vascular Trunks* :

1. Those affecting the *Aorta*.

a. There may be a single arterial trunk, which may be regarded as an aorta sending off branches from different points to the lungs.

b. The aorta may be a vessel from which only the branches of the upper half of the body (and not all of these) are given off, whilst the pulmonary artery, through the ductus arteriosus, constitutes the descending aorta.

c. There may be different degrees of obstruction of the aorta, which may be either very narrow or quite closed from its origin to its point of junction with the ductus arteriosus, in which case it is supplied by the latter with blood from the pulmonary artery. The whole arterial trunk with its ramifications,—in short the whole arterial system,—is in this case frequently disproportionally narrow.

d. The aorta may originate from both ventricles, owing to a deficiency in the partition-wall between them, in which case it is deflected somewhat to the right. The pulmonary artery

is, in this case, either normal, or, as is frequently observed, it is obstructed, narrow, and even closed. The ductus arteriosus, if it be present, then remains open, and carries the aortic blood to the pulmonary artery.

2. Those affecting the *Pulmonary Artery*.

a. The trunk of the pulmonary artery is not only absent where the lungs are wanting, but even where these are present and are furnished with vessels from the aorta.

b. Obstruction of the pulmonary artery, which may be either too narrow or wholly closed, in which case the blood is conveyed to it through the ductus arteriosus from the aorta. This occurs when the right ventricle is imperfect, when the conus arteriosus ends in a *cul de sac*, and very commonly when the aorta originates from both ventricles.

To the above we may add—

3. Anomalies of the *Ductus Arteriosus*.

a. It is sometimes wholly absent.

b. Besides sending several vessels to the head and upper extremities, as do also the branches of the pulmonary artery, it supplies the aorta descendens, or rather merges into it. In this case the aorta diminishes in calibre after giving off its branches, and merges as a thin vessel into the large ductus arteriosus after the latter has been curved into an aorta descendens. (Kilian.) This anomaly occurs either separately and independently, or conjointly with other anomalies of the vascular trunks and of the heart.

c. The most common anomaly is a defective involution of the ductus arteriosus after birth; it either remains open or even in some cases experiences a dilatation. It is, however, much more rarely open than the foramen ovale, whose patency is in general a necessary consequence of different anomalies of the heart and vascular trunks, although even here many exceptions present themselves. On the one hand, it may remain open without any palpable anomaly of the heart and vascular trunks, and on the other it may be contracted, and even closed, where such anomalies exist.

Its involution may be hindered either by the pulmonary artery or the aorta; thus it may either be entirely patent, or may exist as a mere opening closed in the direction of the pulmonary artery, but patent towards the aorta. To this class

belong those cases in adults, where the ductus arteriosus forms a sac-like appendage to the aorta, and where the obliterated ostium arteriæ pulmonalis has the appearance of having been re-opened by violence.

When the ductus arteriosus remains patent, many causes combine to keep the foramen ovale open.

Finally, we also observe a deficiency in the formation of the *valves*. The auriculo-ventricular valves present various obstructions and malformations, generally in connection with a simultaneous malformation of the corresponding ventricle, or associated with a contraction or occlusion of the opening, and principally on the right side. In reference to the arterial valves those of the pulmonary artery are occasionally absent, while malformation and closure of the latter artery, a blind termination of the conus arteriosus, and an abnormal condition of the whole of the right ventricle, are simultaneously present. These valves not unfrequently assume an abnormally inflated annular form, and in some cases only two instead of three valves are observed in the pulmonary artery or in the aorta.

The valve of the foramen ovale is either wholly absent or, as we have already remarked, is imperfect in various ways. In rare cases the Eustachian valve has been found wanting. Its defective involution after birth is interesting, inasmuch as it gives rise to the imperfect closure of the foramen ovale. The Thebesian valve has been found wanting in a few cases.

Malformations *per excessum* affect the heart and vascular trunks in various modes and degrees, and may be referred either to a duplication, or to an arrest of development.

To the *former* belong—

Complete duplication of the heart, or the occurrence of two separate hearts in two distinct pericardia, or in one common pericardium, which is not unfrequent in double monsters, especially where there is duplication of the upper half of the body, while the observations on record regarding a double heart in a single body, are very few in number, and of doubtful character.

Duplication of one or more portions of the heart, or the presence of supernumerary, more or less perfectly separated cavities from which numerous vessels proceed, is of very rare occurrence when there is only a single body. We observe, however, occasionally, in normally formed individuals, a ru-

dimentary partition projecting, in the form of a band or amorphous mass of muscle, into one of the cavities of the heart.

A large heart, from which proceed double vessels, is found in double monsters.

To the *latter* belong—

The persistence of a double aorta ascendens, a cleft condition of that vessel, the persistence of a ductus arteriosus on the right side (Breschet), and the duplication of the upper as well as the lower vena cava.

The valves also occasionally exhibit an excess of formation; we have either an increase of the apices of the auriculo-ventricular valve of the right side, or supernumerary valves, or a multiplication of their apices, with perforation of the ventricular partition; or there may be four semilunar valves in the aorta or the pulmonary artery, or duplication of the Thebesian valve, &c.

§ 2. *Anomalies of Form.*

These are of comparatively common occurrence, and may affect either the external form of the heart, or its internal arrangement; occasionally both varieties are simultaneously present, in which case the deviation from the normal type in the external form is dependent on the anomaly in the internal structure. These anomalies are, moreover, either *congenital* or *acquired*, the former comprising, more especially, original malformations depending upon arrest of development; and the latter, the numerous and various deviations of form developed at different periods of life, and even in the fœtus, as consecutive anomalies, arising more particularly from hypertrophy and dilatation.

The most important original anomalies in the external form of the heart, are combined with and dependent on the already described important anomalies of the internal structure and of the vessels; the more unimportant may be present, associated with very trifling internal anomalies, as for instance, the patency of the foramen ovale, or even, where none of these exist, and where the internal structure is perfectly normal. To this class belong the retraction of the heart's apex—apex cordis bifidus, an arrest at an early stage of development—and the rounding of the apex of the heart, associated with predominant width of the whole heart,—an arrest at a very advanced period of development.

This last named form, which depends on an equality in the size and thickness of both ventricles, is often continued to late periods of extra-uterine existence, and is maintained, together with the simultaneous patency of the foetal passages, by the defective development of the lungs, whose proper functions may be mechanically obstructed by the form of the sternum, as is observed in rachitis.

Among the unimportant and incidental anomalies depending, very frequently, on various degrees of contraction and rigor, we must reckon different forms of the heart which approximate to the round type, and are either long, slender, wedge-shaped, pad-like, spirally curved, broad and obtuse, &c.

In order to avoid repetition, we would refer our readers to the sections on hypertrophies, dilatations, and textural diseases, for further and special notice of the *acquired* anomalies of the heart.

§ 3. *Anomalies of Position.*

These are either *congenital and original*, or *acquired*. The former are very numerous, and admit, in part, of being referred to an arrest of development. Many depend on different adhesions of the heart resulting from inflammation in the foetus, and some again on different anomalies of neighbouring organs, as, for instance, on the deficient development of a lung, the partial deficiency of the diaphragm, and the position of the abdominal viscera in the thorax. These anomalies are very various in their character, and the most important, together with the vascular anomalies included in this class, are as follows:

Position of the Heart exterior to the Body.—This anomaly occurs associated with a partial absence of the diaphragm and the abdominal and thoracic walls. Where the former of these is absent, the heart is generally situated with all or several of the viscera externally to the body, in a closed or open sac occasionally contained in the sheath of the umbilical vessels.

Position of the Heart within the Body, but external to the Thoracic Cavity.—According to the direction in which the heart is placed, it assumes either a cervical position, (*ectopie cephalique*,) or an abdominal one (Breschet).

Anomalous Positions of the Heart in the Thoracic Cavity.—

These possess various points of interest from their presenting considerable analogy with many acquired anomalous positions of the heart, and also on account of their apparently arising from similar conditions. To these belong the position of the heart on the right side, without the simultaneous transposition of other viscera, its perpendicular position in the centre of the thoracic cavity, its horizontal, oblique positions, &c.

Anomalous Origin of the Vascular Trunks.—To this class belong:

The displacement of the aorta towards the right side, and its origin from both ventricles, associated with a defect in the ventricular septum; or the aorta may take its origin, conjointly with the pulmonary artery, from the right ventricle, where no such anomaly exists. We sometimes find a similar relation of the pulmonary artery, that is to say, it takes its origin from both ventricles, or conjointly with the aorta from the left ventricle. This vessel has also been observed to spring from abnormal positions in the right ventricle.

Many anomalies of the systemic and pulmonary veins, as, for instance, the opening of a left descending vena cava into the auricle, an opening of the pulmonary veins of the right side into the right auricle, into the upper vena cava, &c., also belong to this class.

Actual transposition may exist with reference to the heart alone, or conjointly with the thoracic and abdominal viscera generally. A more important transposition is, however, that affecting the vascular trunks, which often present the anomaly of the aorta springing from the right, and the pulmonary artery from the left ventricle, while the veins open normally. Otto found, in the case of a double monster, that the venæ cavæ opened into the left, and the pulmonary veins into the right auricle, while the arterial trunks presented the normal mode of origin.

The acquired changes of position of the heart are very numerous, but as they are merely secondary phenomena, they generally possess a very subordinate interest. An exception occurs, however, in the case of those anomalies of position of the heart, which arise from empyema, pneumothorax, pulmonary emphysema, atrophy of the lungs, &c., and which are of great importance with regard to diagnosis.

The majority of these consist in a displacement of the heart from its normal position. It may occur in the most opposite inclinations either to one or the other side, or downwards, upwards, forwards, or backwards. The most common causes of these displacements are, on the one hand, excessive dilatation of one or other of the pleural sacs from exudations into its cavity, from pneumothorax or pulmonary emphysema; and on the other, the formation of a vacuum in it by the cure of chronic pleurisies, by wasting and atrophy of the lungs consequent on indurated pneumonia, by bronchial dilatation, &c. The displacements of the heart towards one or the other side resemble similar congenital and original anomalies of the heart's position. A change in the position of the heart is but rarely occasioned by pneumonic and tuberculous enlargements of a lung, and still more unfrequently by an acquired position of the abdominal viscera in one side of the thorax, arising from laceration of the diaphragm, &c. The heart may likewise be differently displaced from its position by aneurisms of the aorta, the contiguity of voluminous adventitious products, &c. Flatulence, extreme ascites, and large adventitious products in the abdomen, may also displace the heart in an upward direction; whilst a corresponding anomaly in the position of the heart is likewise induced by curvature of the spine, irregularity in the form of the thorax, &c.

In contrast with the above named anomalies, this change of position may sometimes be *spontaneous*, in consequence of the heart assuming an anomalous position and anomalous relations of contact with the diaphragm and ribs, arising from an uniform or a varying enlargement, and from its simultaneous increase in weight.

§ 4. *Anomalies of Size.*

These anomalies manifest themselves either by an *abnormal excess or deficiency of size*. Both conditions may be either congenital or acquired, and are of great importance from their frequent occurrence and the serious and numerous consecutive disturbances to which they give rise.

In order to arrive at a correct opinion regarding an individual case, it is especially necessary for the student to acquire a knowledge of the normal size of the heart and of its individual

portions. Many measurements have been made in recent times, and from these a mean or average standard has been deduced.

The results yielded by Bizot's measurements appear to us to be most correct. We will limit ourselves to the dimensions in adults, between the ages of 30 and 49 years.

The following are the mean measurements:

| | <i>In Men.</i> | | <i>In Women.</i> | |
|---|-------------------|-------------|-------------------|-------------|
| The length of the heart | 43 $\frac{3}{25}$ | Paris lines | 41 $\frac{2}{27}$ | Paris lines |
| „ breadth „ | 47 $\frac{1}{25}$ | „ . . | 44 $\frac{1}{27}$ | „ |
| „ thickness „ | 17 $\frac{3}{25}$ | „ . . | 14 $\frac{5}{27}$ | „ |
| „ length of the left ventricle . | 29 $\frac{1}{25}$ | „ . . | 31 $\frac{1}{27}$ | „ |
| „ breadth „ „ | 53 $\frac{3}{25}$ | „ . . | 46 $\frac{4}{27}$ | „ |
| „ length of the right ventricle . | 37 $\frac{1}{25}$ | „ . . | 33 $\frac{3}{27}$ | „ |
| „ breadth „ „ | 83 $\frac{1}{25}$ | „ . . | 76 $\frac{1}{27}$ | „ |
| The thickness of the walls of the | | | | |
| left ventricle | | | | |
| „ „ at the base | 41 $\frac{7}{25}$ | „ . . | 41 | „ |
| „ „ in the middle | 51 $\frac{1}{25}$ | „ . . | 43 $\frac{1}{27}$ | „ |
| „ „ near the apex | 31 $\frac{3}{25}$ | „ . . | 31 $\frac{6}{27}$ | „ |
| The thickness of the interventricular | | | | |
| septum in the centre | | | | |
| „ | 42 $\frac{1}{25}$ | „ . . | 41 $\frac{1}{27}$ | „ |
| The thickness of the walls of the right | | | | |
| ventricle | | | | |
| „ „ at the base | 13 $\frac{3}{25}$ | „ . . | 11 $\frac{6}{27}$ | „ |
| „ „ in the middle | 12 $\frac{7}{25}$ | „ . . | 11 $\frac{1}{27}$ | „ |
| „ „ near the apex | 11 $\frac{1}{25}$ | „ . . | 9 $\frac{6}{27}$ | „ |
| The width of the auriculo-ventricular | | | | |
| openings | | | | |
| „ „ of the left | 48 $\frac{2}{25}$ | „ . . | 40 $\frac{1}{27}$ | „ |
| „ „ of the right | 54 $\frac{5}{25}$ | „ . . | 47 $\frac{4}{27}$ | „ |
| The width of the origin of the aorta | | | | |
| (above the valves) | | | | |
| „ | 30 $\frac{3}{25}$ | „ . . | 28 $\frac{1}{27}$ | „ |
| The width of the origin of the | | | | |
| pulmonary artery | | | | |
| „ | 31 $\frac{1}{25}$ | „ . . | 29 $\frac{1}{27}$ | „ |

We purpose, in the sequel, making further use of the other results of the labours of Bizot, which possess any degree of interest, and are not opposed to our own observations.

According to Bizot, the heart increases in volume from birth to extreme age; this increase being most considerable to the age of 29, after which it is only appreciable by measurement. Augmentation of volume depends especially on the continuous dilatation of the openings, and on the increase of thickness of the walls of the ventricles, which is always most strongly marked in the case of the left, and is indeed scarcely perceptible in that

of the right ventricle. The dilatation of the auriculo-ventricular openings is tolerably uniform, and that of the arterial equally so until middle life, but after that period the opening of the aorta is more rapidly dilated than that of the pulmonary artery, the latter becoming even narrower than the aorta. In children both the arterial openings remain equally wide, till from the sixth to the tenth year. The cavities of the right side of the heart have a greater capacity, and their openings are wider.

Bizot's opinions regarding the influence of sex and bodily frame are, that the dimensions of all the parts collectively are smaller in women than in men; that the auriculo-ventricular openings in particular are narrower, whilst the opening of the pulmonary artery is relatively wider in them than in men. In tall persons of either sex, the heart, according to the same authority, is relatively smaller than in persons of shorter stature, while it is larger in broad than in narrow-shouldered persons.

We adopt Bouillaud's data for the walls of the auricles, with a remark, however, that his estimate is too high; according to him, the thickness of the wall of the left auricle is one Paris line and a half, while that of the right auricle is one line.

Laennec proposed to establish a scale for the relative measurement of the size of the heart, and rejected as inefficient all data of the weight and size of the heart that had been obtained without regard to individual bulk. The basis on which he founded his conclusions was, that the heart, including the auricular appendages, should be of a volume equal to that of the fist of the individual, or only in a slight degree either larger or smaller. The walls of the left ventricle should be somewhat more than twice as thick as those of the right one; the left ventricle, when cut open, should remain unclosed, while the somewhat wider right ventricle, which, notwithstanding the thinness of its walls, is furnished with more considerable trabeculae, should collapse.

If in the consideration of these data, (which are, however, many respects defective,) regarding the relative thickness of walls of the right and left ventricles, we bear in mind that dilatation is not only very commonly, according to Andral's opinion, as 1:3, but still more frequently (see Bizot's data), and that these measurements refer only to middle life,

the statements the columnae carneae are not taken into consideration.

we shall find that Laennec's comparison of the size of the heart with that of the fist deserves considerable attention, remembering that it must be received as simply approximative, and limits itself to cases where there is no apparent disproportion in the size of the fist. We have, therefore, as a general rule, regarded the heart as of a relatively normal size, when it was equal to that of the fist, and when there was an absence, both during life and after death, of any indications of cardiac disease.

The weight of the heart, in its normal condition, has been variously estimated, and may be from eight to ten ounces. (Compare Lobstein, Bouillaud, and Cruveilhier.)

A. *Abnormal size.*

Deviations in the size of the heart depend either on *hypertrophy* of its muscular substance (augmentation of its mass) or on *dilatation* of its cavities; while both conditions, with a preponderance of the one or the other, very commonly constitute the basis of the higher degrees of enlargement of the heart, as we shall presently have occasion to show.

Here, as has already been indicated, we will merely consider hypertrophy of the muscular substance. There is, however, a form of disease, for which we know no better denomination than *hypertrophy of the endocardium*, and of which, however much it may seem to belong to the subject under consideration, we will treat subsequently in connection with endocarditis,—a disease with whose products it may easily be confounded, and for which it undoubtedly is frequently mistaken. We will also defer the consideration of *hypertrophy of the valves* to a subsequent portion of our work.

a. Hypertrophy of the muscular substance of the heart (*hypertrophia cordis*) constitutes either *total* or *partial hypertrophy*, as it affects the whole, or only some portions of the heart, and is characterised by various degrees of intensity.

Total hypertrophy is, in most cases, so far unequal that it usually preponderates in one section of the heart,—commonly, although not invariably, in the left portion,—where it forms the starting-point of a morbid development of bulk.

Partial hypertrophy affects either the whole of one of the larger sections of the heart, as for instance the walls of one of the cavities, or it attacks only certain parts of that section. Thus

it is very frequently limited to the true muscular wall of one ventricle, thickening it in various degrees, while the papillary muscles and the trabeculae retain their normal volume, or even where they have become perceptibly thinner and fainter, simultaneously with the dilatation of the affected cavity. In other cases hypertrophy principally attacks the papillary muscles and the trabeculae, whilst the true fleshy wall is only moderately increased in thickness. The former of these conditions occurs generally in the left; and the latter, that is to say, an excessive development of the trabeculae, in the right ventricle.

Hypertrophy of the muscular wall of a ventricle may present great variations; it may, in one case, affect the whole, in another it may be limited to one portion of the ventricle, as for instance the base, the middle part or the septum, or again it may predominate in one or other of those sections.

Hypertrophy of the auricles is generally uniformly diffused over the wall, but it occasionally preponderates in the atrium towards the appendage.

The degrees of hypertrophy present still more numerous differences.

If we follow Bizot's data we must assume that ventricular hypertrophy is present when the thickness of the muscular wall of the left ventricle in men is about 6''' (Paris measure), and in women is about 5'', and when the right ventricle in men is about 3''' in thickness, and that in women 2½''' in thickness. From this point hypertrophy may pass through every varying degree, till it induces so enormous an increase of bulk, that the walls of the left ventricle attain a thickness, varying from an inch to an inch and a half; that the walls of the right ventricle vary from 6 to 9 lines, that the walls of the left auricle vary from 2 to 3 lines, or even more, and that the walls of the right auricle vary from 1½ to 2 lines.

The weight of an hypertrophied heart may range from one to two pounds, and even higher.

The most important and serviceable classification of hypertrophies of the heart is that which is based on a reference to the condition of the cavities of the heart, more especially in regard to their capacity. (Bertin, Bouillaud.)

1. The capacity of the hypertrophied portion of the heart

may remain normal, constituting *simple hypertrophy*, in which the dimensions of the heart are increased.

2. The cavity of the heart may be dilated, constituting *eccentric hypertrophy* (*hypertrophia excentrica, centrifuga*); here also the dimensions of the heart are increased.

3. The cavity of the heart may be contracted, constituting *concentric hypertrophy*, (*hypertrophia concentrica, centripeta*.) The dimensions of the heart may here be increased, normal or diminished.

We shall treat more fully of these different forms when we take into consideration the most essential points relating to dilatation of the cavities of the heart.

b. The dimensions of the heart are more increased by the dilatation of its cavities than by hypertrophy. This dilatation, (*dilatatio cordis*, also *aneurysma cordis* in the older writers,) may, like hypertrophy, be *total*, affecting all the cavities of the heart, or *partial*, attacking only one of these portions. The excessive degree which dilatation may attain led the early anatomists to compare the human heart to that of a bullock.

Here also the most useful classification is that which is founded on the relation of the walls of the diseased portions of the heart.

1. Dilatation of the cavities of the heart may occur conjointly with hypertrophy of their walls, constituting *aneurysma cordis activum* (Corvisart), and with *eccentric hypertrophy*.

2. Dilatation of the cavities of the heart may exist, associated with walls of normal thickness. This condition of simple dilatation—*aneurysma cordis simplex*—deserves equal attention with the above named form of *dilatation with hypertrophy*, in as far as the normal thickness of the walls of the heart in dilatation of the cavities must necessarily depend on hypertrophy; it may therefore be regarded as an *active dilatation*—*aneurysma cordis activum*.

3. Dilatation of the cavities of the heart may occur in combination with attenuation and relaxation of the walls, constituting *passive dilatation*—*aneurysma cordis passivum*. (Corvisart.)

Besides these forms of dilatation, there are others connected

with and dependent on alterations of texture, limited to one portion of a single cavity, and which we will pass over in the present place, reserving their consideration for a more suitable occasion, under the head of inflammations of the heart. We would here merely observe, that these forms of dilatation are known as *partial dilatation* and *partial aneurism of the heart*. As, however, we have applied the former of these terms to dilatation of a single division of the heart, and as, on the one hand, the term aneurism is unsuitable to the forms of dilatation under consideration, while, on the other, dilatations, combined with and dependent on alterations of texture, and such as attack only one portion of a cavity of the heart, exhibit great affinity with aneurisms of the arteries, we would designate these last named, which we pass over for the present, as true *aneurism of the heart*.

On considering the above named forms of hypertrophy and dilatation, we find *five*, or perhaps more correctly speaking, *four different conditions*, and these will constitute the subject of the following remarks :

Simple hypertrophy, which is in general of rare occurrence, affects the ventricles, attacking the left one more frequently than the right. It probably continues to exist only for a certain period of indefinite duration, and then gradually merges into eccentric hypertrophy, that is to say, hypertrophy with dilatation. Although scarcely a doubt can be entertained of its existence, the attempt to confirm it is not devoid of difficulty.

The existence of *concentric hypertrophy* has been doubted by many who have made observations on the human subject after death, and prosecuted experiments on animals. In those who die from loss of blood, and occasionally after sudden and violent modes of death, the heart is indeed often in a condition of contraction, which might easily be mistaken for concentric hypertrophy. We cannot, however, agree with those who doubt the existence of concentric hypertrophy, which occurs, although rarely, in both ventricles, and, according to our observations, more frequently in the left. The cavity of this portion of the heart appears contracted in consequence of the thickening of the muscular wall, and of the papillary muscles, and the trabeculæ. Disease of the heart is manifested during life, and

the symptoms exhibited correspond to the appearances observed after death.

Eccentric hypertrophy, active dilatation, including *simple dilatation*, is incomparably the most frequent condition. It attacks the ventricles as well as the auricles, and most frequently the cavities of the left side. Active dilatations originate in one portion of the heart, and, beginning at the left ventricle, gradually extend over the whole organ. This condition gives rise to the highest degrees of cardiac enlargement, which were known to the ancients under the terms *enormitas cordis*, *cor taurinum*, &c. The enlargement is most strikingly manifested at the conus arteriosus of the right ventricle, whilst the space of the actual ventricle is generally contracted by the intrusion of the arch of the septum. The auricles are occasionally the special seat of active dilatation, and in these cases the disease commonly depends on contraction of the auriculo-ventricular opening on the same side. The wall of the auricle is stiff and rigid, and the cavity is not unfrequently filled with coagulated blood, or occasionally with stratified coagula of fibrin. These forms of dilatation have, however, been observed unassociated with this form of contraction.

Passive dilatation, in its lesser degrees, is of frequent occurrence. It attacks the ventricles as well as the auricles, especially the cavities on the right side of the heart, and the right auricle most frequently. When it affects the left ventricle it is most commonly and most decidedly seen at its apex, where it first manifests itself. Intense degrees of this form of disease are unusual, although the auricles in particular are capable of remarkable dilatation.

These different forms may be variously combined. Where disease has attacked the whole of the cavities of the heart, one cavity is usually disproportionally affected beyond the others, and heterogeneous forms are observed to arise and exist in conjunction with one another, as for instance hypertrophy and active dilatation of the left side, together with passive dilatation of the right. In most cases the disease predominates in that portion of the heart's cavity which was first and, from some obvious cause, most intensely affected. Such is, however, not invariably the case, since consecutive disease occasionally super-

venes, which fully equals or even exceeds that in the cavity originally attacked.

Dilatation of the openings usually exists in conjunction with dilatations of the heart generally, corresponding in intensity with the various degrees of the latter, and depending most probably on one common cause. In active dilatations, the arterial openings are more prominently affected, whilst in passive dilatation the auriculo-ventricular openings more frequently participate in the disease. In this form of dilatation the valvular apparatus very commonly remains sufficient, in consequence of an enlargement of the valves, attended by a striking attenuation and an elongation of the tendons of the papillary muscles.—We must, however, be careful not to confound these forms of dilatation with dilatation of the commencement of the aorta, which is of very frequent occurrence, and depends on a diseased condition of its coats, for the latter will occasion dilatation of the left side of the heart, with a frequency proportional to the association of dilatation of the vessel with insufficiency of its valves.

It is important to notice that there is a relaxed condition of the heart after death, which is very similar to *passive dilatation*. In the rapidly decomposing bodies of those who have died of acute dyscrasæ, the heart is very commonly collapsed, visibly dilated, easily torn, and characterised by thinness of the walls, various decolorations of the muscular substance, and imbibition of hæmatin in the endocardium and along the coronary veins. It is very probable that a similar condition of the heart manifests itself in every case at a certain period after death. The above named requirements of its occurrence enable us to recognise this phenomenon as the result of decomposition, but the difficulty attending its diagnosis in the dead body reminds us of that which attaches to the question of the existence of concentric hypertrophy in so far as this condition is undoubtedly very frequently to be referred to the *agonia mortis*.

In *simple dilatation* we also occasionally meet with a condition of the muscular substance of the heart, which gives it a passive character.

In cases of *active dilatation (eccentric hypertrophy)* the trabeculæ are frequently so completely atrophied as not only to

be attenuated by elongation, but even entirely severed, their existence being indicated along the greater and middle part merely by the inner cardiac investment surrounding them, and by the muscular substance of which their terminations are composed.

We are not yet able to explain why, under analogous or very similar conditions, dilatation of the heart will be developed in one case in a passive, and in another in an active form. We will append to our enumeration of the causes of these diseases of the heart the form of disease that is usually dependent on each, merely remarking here, in general terms, that, in our opinion, considerable mechanical obstructions generally, and sometimes with great rapidity, induce an excessive degree of dilatation, whilst, on the other hand, lesser and more slowly developed obstructions give rise to hypertrophy.

The *form* of the heart undergoes various alterations in consequence of these enlargements. Its malformation is the more important in proportion to the enlargement, and the more it is confined to, or preponderates in one single cavity of the heart. It affects the external as well as the internal form. In simple, and still more manifestly in eccentric hypertrophy (active dilatation) of the left ventricle, where the chief seat of disease is at the base and the middle portion, the heart assumes a round wedge-like form, while in the more advanced stages of the disease, the whole ventricle is swelled into a pad-like shape. The malformation which especially consists in dilatations of the left ventricle, expands towards the right ventricle, into which the septum is bent in an arched form; its space being so considerably contracted, that it appears like a mere appendix to the heart, while its conus arteriosus appears dilated and hypertrophied. Dilatations of the right ventricle widen the heart at its base, and from thence down to the apex. Where there is simultaneous dilatation of the left ventricle, the heart acquires the form of an obtuse triangular pyramid, or a discoidal mass. Active dilatations of the conus arteriosus of the right ventricle, which are of frequent occurrence, lead to malformation of the heart by enlarging its circumference near the base, &c.

The *position* of the diseased heart becomes the more anomalous in proportion to the volume and weight which it acquires. In a slight degree of enlargement, the heart inclines less to the

left side of the thorax, while in excessive forms of enlargement and dilatation, it has its base almost diagonally inclined to the right and its apex to the left side, whilst its right half rests on the anterior thoracic wall, contracting both thoracic cavities in the region of the lower lobes of the lungs, and causing them to press in one large surface on the diaphragm, which is thus more or less pushed downwards on the epigastrium.

The *colour*, *consistence*, and *texture* of the muscular substance of the diseased heart, present numerous differences.

The colour of the *hypertrophied heart* is most frequently dark, and of a brownish red hue; the consistence is generally greatly increased, and the texture apparently normal. It must here be remarked that the consistence of the right ventricle presents a striking anomaly in the more highly developed forms of hypertrophy, the texture acquiring a toughness which is never observed under any condition in the left ventricle. The walls which become rigid and retract on being cut, exhibit extreme resistance and hardness, and yield, when struck, a sound which, according to Laennec, resembles the tone emitted from hard leather. A similar relation is observed in active dilatation of the auricles, when excessively hypertrophied. This increase of consistence seems to depend on the deposition of a great quantity of organic matter in the form of a finely granular substance, and in the production of new flat muscular fibres without transverse striae.

In other, and very frequent cases, the hypertrophied tissue of the left ventricle presents another character. Its colour appears to be faded, and of a dirty brown or yellow tint, either in separate points in the form of foci, or over a layer, generally an internal one, whose thickness varies, or finally throughout the whole thickness of this portion of the heart. The consistence then becomes modified in a peculiar manner, the walls of the heart become rigid, tough, and capable of resistance, while their tissue loses its proper firmness, is fragile, and easily broken down. The texture is perceptibly altered, although in what manner the change is effected is not known. According to our investigations, this disease of texture must be regarded as a form of morbid fatty degeneration of the heart, similar to that treated of under Form 2 of Diseases of the Muscles; and we will therefore consider the subject more at large

under the head of anomalies of texture. We are moreover of opinion, that it associates itself with hypertrophy as a consecutive disease; that is to say, that after being once developed in the hypertrophied tissue, it favours the dilatation of the hypertrophied portion of the heart, and very frequently gives rise to those spontaneous ruptures which occur in this organ.

Traces of inflammation not unfrequently occur, either with or without the above named alterations of texture in the muscular substances of the left ventricle, when it is the seat of active dilatation. One or more points or foci of limited extent, either on the surface or lying deep in the texture, occasionally exhibit a redness and injection of the bleached and flabby tissue, which is infiltrated with gelatinous, fibrinous, or purulent matter. More frequently these are the residua of a former inflammation,—spots at which we find the muscular substance replaced by a white ligamentous (fibroid) texture. (See the section on ‘Inflammation of the muscular substance of the Heart.’) These latent and recurring processes of inflammation are in some instances connected with the residua and secondary effects of pericarditis and endocarditis, and undoubtedly would appear to promote the origin and further development of cardiac disease.

In *passive dilatations*, the colour of the tissue of the heart is occasionally purplish red, but more frequently it is darker and bluish red, owing to the imbibition of the hæmatin, which is greatly favoured by the dissolved condition of the blood, and the relaxation of the whole tissue. The muscular substance of the heart is in these cases extremely flabby and easily torn, while its walls collapse when they are cut open. In the higher stages of dilatation the muscular bundles in the auricles are forced asunder, so that the wall of the heart appears between them as a mere membrane.

In dilatations arising from pericarditis, the muscular substance has a dirty rusty brown, or yellow leather-like colour, is easily torn, and appears as if half boiled; in other cases, it is pale, flabby, and abnormally fat, the surface of the heart being frequently covered by an accumulation of adipose matter.

The knowledge of the *causes of the origin of these diseases of the heart* is of the highest interest. Many admit of being discovered and made apparent without any great difficulty, but

many others are partly problematical and partly uninvestigated. We will consider them in such an order as to proceed from those which are obvious to those which are less apparent, and finally to the problematical and hypothetical, giving special attention to the practically important ones comprised under each category.

These causes are as follow:

1. *Mechanical Obstructions*, which give rise, according to circumstances, either to preponderance of dilatation or preponderance of hypertrophy.

a. *Mechanical Obstructions in the Ostia of the Heart*.—The number of diseases produced by these causes is probably the greatest. They are consequent on various diseases of the arterial and auriculo-ventricular valves, more especially on the secondary effects of endocarditis (Bouillaud's chronic endocarditis), and admit of being generally referred to *contraction (stenosis) of the ostium*, and to *insufficiency of its valves*. Whether the latter has a tendency to give rise more frequently to dilatation, and the former to hypertrophy, has not yet been determined.

As these diseases of the valves are far more frequent in the left than in the right side of the heart, so also are the diseases of the heart to which they give rise. The auricle or the ventricle becomes the more acutely affected according to the seat of the valvular disease, while both are simultaneously attacked where the arterial and auriculo-ventricular valves are alike diseased. Owing to the impediment presented to the emptying of the heart's cavities on the left side, and the consequent obstruction of the capillary circulation through the lungs, disease extends to the right ventricle, and from thence to the right auricle, commonly manifesting itself as hypertrophy with excessive dilatation, and occasionally—more especially in the auricle—as passive dilatation.

b. *Mechanical Obstructions in the Arterial Trunks*, occurring at different distances from the heart, promote the development of cardiac disease in proportion to their greater vicinity to that organ.

These obstructions are of various kinds.

Congenital contraction of one or other of the vascular trunks is not unfrequent, and is generally manifested in the aortic trunk by an insufficiency of calibre which extends to the

branches, and probably also to the more delicate ramifications. This condition induces very considerable dilatation with more or less hypertrophy in the left ventricle, and subsequently leads to similar dilatations of the left auricle, and of the cavities on the right side of the heart.

To this class belong acquired contractions of the vascular trunks and their main branches, together with their final obliteration, dependent on alterations of texture.

Further, the contractions caused by compression or expansion, and the obstructions presented to the current of the blood by the elongation, angular curvature, twisting, &c., of the large arteries.

Lastly, there are also dilatations of these arterial trunks, which, as is well known, appear frequently, and in the most excessive degrees, in the trunk of the aorta and its main branches, under the various forms of aneurism. These conditions generally induce active dilatation of the left ventricle, with a rapidity and intensity proportional to their vicinity to the heart and to their importance. It must be observed, that although insufficiency of the valves of the diseased trunk may simultaneously occur, its existence is not necessary to the formation of cardiac disease, which is then owing to the obstacle opposed to the advance of each successive blood-wave by the mass of blood accumulated in the dilated and paralysed trunk.

c. Similar (mechanical) Obstructions in the Capillaries.—Obstructions in the capillaries of the pulmonary artery are tolerably evident, inducing active dilatations of the right ventricle, and, subsequently, dilatation of the right auricle, having in some cases a more active and in others a more passive character. To these belong:

a. The obstruction presented to the circulation through the capillary system of the lungs by contraction of the thoracic cavity, and the consequent excessive thickness of the pulmonary texture. This condition presents a great degree of intensity and a character of constancy in malformations of the thorax and contractions of its cavities consequent on curvature of the spine, more especially extreme scoliosis and kyphosis, and in cases of rachitic chicken breast. When the above named diseases of the right side of the heart attain a great degree of

intensity, they commonly give rise to highly developed hypertrophy.

β. Next, we must notice an increased condensation and atrophy in a more or less considerable portion of the lung, in consequence of compression from pleuritic exudation and of its healing, and of indurated pneumonia. The development of the heart will be proportional to the actual atrophy of the pulmonary texture, to the extent of the surface atrophied, and to the degree in which the capillaries have been destroyed by obliteration.

γ. Atrophy of the pulmonary texture associated with extended and considerable bronchial dilatation.

δ. The obstruction presented to the circulation through the capillaries of the lungs by their emphysema (emphysema vesiculare.) This condition depends at first on the continued excessive expansion of the pulmonary cells, and in the more advanced stages on the obliteration of the capillaries which occurs in an uniform degree with the atrophy of the texture of the lung. The importance of the heart-disease depends on the extent, degree, and period of duration of the emphysema; occasionally the disease is very intense.

ε. The obstruction opposed to the injection of the lungs by the pulmonary artery, owing to the insufficient emptying of the pulmonary veins in consequence of disease of the left side of the heart, and of the habitual over-filled condition of the capillaries of the lungs. Hence arise the greatest number of diseases of the right side of the heart, and all the numerous consecutively developed dilatations of the right ventricle and auricle, which originate in the left cavities, and are extended by means of the capillaries of the pulmonary system. The most important of these forms of dilatation are those which, in the manner already indicated, depend upon a contraction of the auriculo-ventricular opening on the left side of the heart.

It is only in comparatively rare cases that tuberculosis of the lungs and tuberculous pulmonary phthisis give rise to even a very moderate degree of active dilatation of the right side of the heart. A certain degree of diminution in the size of the heart is more frequent, and is then manifested in the form of simple or even concentric atrophy, which corresponds with the general tabes and the wasting of the mass of the blood.

On the other hand, similar obstructions in the *capillaries of the aortic system* are either wholly unknown, or are so obscure, that although *à priori* conjectures may be hazarded regarding their persistent or transient existence, no physical (anatomical) demonstrable facts can be established in reference to the subject.

2. *Diseases of the Texture of the Heart.*—To these belong :

a. First, and most prominently, *inflammations*, as for instance of the pericardium, the muscular substance of the heart and the endocardium, both in their primary and secondary character. By paralysing the substance of the heart, inflammations occasion dilatations, which are maintained by their own secondary conditions, which mechanically augment them, and gradually superinduce hypertrophy.

An important place must be assigned to dilatations arising from chronic pericarditis, especially when associated with purulent exudation, or when investing the heart with a pseudo-membrane incapable of contraction, or, lastly, when there is firm adhesion of the heart to the pericardium consequent on these new structures. Inflammation, in proportion to its intensity, and the quantity and purulent character of its effusion, tends to promote paralysis of the muscular substance of the heart, accompanied by decoloration and diminished cohesion, and hence furthers the development of passive dilatation. The longer the inflammation has continued, the more permanent will be the character of the cardiac affection, and if at length the heart adheres to the pericardium, a mechanical obstruction is opposed to the contraction of the former by the pseudo-membrane, which agglutinates the pericardium to the heart. This form of dilatation commonly affects the whole heart.

The dilatations induced in the same way by the endocarditic process, are similar to the former, but are usually less important; and when it gives rise to anomalies of the valves, they may gradually assume the active form by the association of hypertrophy. The left side of the heart, especially the left ventricle, is incomparably the most frequent seat of these affections.

It must be evident that the importance of the dilatation induced by the inflammation of the muscular tissue of the heart, will be in proportion to the frequency of inflammation,

and the number and extent of its starting-points. This form of dilatation, excepting in very rare cases, invariably affects the left ventricle.

b. Adiposity of the Heart.—An excessive accumulation and formation of adipose tissue in the heart promotes dilatation of a passive character, in consequence of the simultaneous attenuation of the muscular walls of the heart.

The form which we regarded as consecutive, in speaking of the condition of the texture of the hypertrophied and dilated heart, appears to favour further dilatation.

3. Finally, in all those cases in which cardiac disease cannot be referred to any of the above enumerated causes, it may originate in excessive *innervation* of the heart. Under this head we may include a considerable number of cases of hypertrophy and dilatation of the left side of the heart, which Bouillaud has termed *primary*, in order to distinguish them from consecutive forms arising from the causes already indicated.

Many of these causes, more especially endocarditis and its secondary conditions, are occasionally observed in the fœtus, and the diseases to which they give rise under these circumstances are then *congenital*. Other cardiac diseases belonging to this category depend on *original malformation* of the heart, its ostia and vascular trunks, and constitute a special series, of which we purpose treating subsequently under the head of cyanosis.

Independently of these causes, the *consecutive diseases*, arising from *affections of the heart*, are alike important and numerous. The following are the most worthy of notice :

Excessive Fulness and Dilatation. Stasis in the whole Venous System.—This condition is most strikingly manifested in the great venous trunks,—the venæ cavæ, and the trunk of the portal vein,—from whence it extends along their branches into the capillaries, and is then characterised by distension and *cyanosis*.

Hæmorrhages, resulting from the excessive fulness of the capillary system, manifested in discharges of blood from the mucous membrane of the nose, excessive menstrual uterine discharge, bleeding from the bronchial and pulmonary mucous membrane (hæmoptysis and hæmoptoic infarctus), hæmorrhage

from the intestinal mucous membrane, from the liver, (apoplexy of the liver,) and into the brain. The most frequent and important of these, are bleeding from the bronchial and pulmonary mucous membrane, and cerebral hæmorrhage; and we will, therefore, treat of them specially, together with other subjects, in a future page.

Hypertrophies, more especially affecting the parenchymatous abdominal viscera, as the liver, spleen, and kidneys, although more particularly the two former. These affections are frequently marked by a visible increase in the volume of the organs, by a persistent tumour, and more commonly—either with or without the former,—by a striking increase of consistence depending on a compression of the elementary structure induced by hypertrophy.

To these we must add *hypertrophies of the mucous membranes and the chronic catarrhal inflammatory conditions—the forms of blennorrhæa*—to which they give rise. Those which are most remarkable for their intensity and extent are bronchial catarrh, and a catarrhal condition of the whole of the intestinal mucous membrane.

Dropsy which usually manifests itself first as anasarca of the lower extremities, and is then converted into general dropsy by the addition of serous effusions into the large serous sacs, is the result of the above named venosity and mechanical hyperæmia. Œdema of the lungs is highly important, whether it be slowly established, and as a result of dropsy in other parts, or whether it show itself among the earliest symptoms of dropsy, and speedily attains a high degree of intensity, when it not unfrequently proves rapidly fatal.

Besides these secondary conditions, there are others which, from their importance, merit special consideration: these are certain *diseases of the liver*.

The diseases of the liver, of which we are about to speak, have frequently been regarded as causes of disease of the heart, but it is not very clear in what manner they can be supposed capable of bringing about such a result. Facts as well as theory tend rather to show, that the morbid condition of the liver is a consequence of heart-diseases, and is developed by the constant mechanical hyperæmiæ induced by the latter. To this class belong, besides the hypertrophy already described,

the condition known as *nutmeg liver*, which is generally developed in a very intense form, that is to say, as a sharply defined saturated yellow substance, very rich in blood, and marked with well-defined red patches; and, finally, that *granular condition of the liver* which is gradually developed from the latter, either with or without inflammation.

In our observations on hæmorrhages, we spoke of *those of the bronchial and pulmonary mucous membranes*, and of *cerebral hæmorrhage*, as among the most important results of the cardiac diseases under consideration. Cerebral hæmorrhage (apoplexia gravis) occurs in so large a majority of cases in conjunction with disease of the heart, that the latter has, with much reason, been regarded as a predisposing cause of cerebral apoplexy. The disease of the heart consists here in simple hypertrophy of the left ventricle, or what is much more frequently the case, in its dilatation, associated with highly developed hypertrophy. Cerebral apoplexy is undoubtedly induced by laceration of the cerebral vessels, occasioned by the augmented impulse propagated from the left ventricle; and this is the more easily effected the more the arterial coats, in advanced life, have lost their normal texture and cohesion, their power of resistance, and elasticity, or have become ossified, &c. A similar or even identical relation has been supposed to exist between hæmorrhages of the bronchial and pulmonary mucous membrane and active dilatation of the right side of the heart. It must, however, be observed in reference to this point, that hæmorrhages of this kind very frequently occur in dilatations and hypertrophies of the most different portions of the heart, in the form of hæmoptysis and hæmoptoic infarctus, (pulmonary apoplexy.) The cases in which they are found to exist, are very rare in comparison with the frequency of active dilatation of the right ventricle, and their coincidence bears a very secondary relation to the frequency of the co-existence of active dilatation of the left ventricle and cerebral apoplexy. Whether this depends on the absence of that diseased condition of the coats of the vessels in the branches of the pulmonary artery which is found to exist in the cerebral vessels, is a point that has not been determined, since, on the one hand, cerebral apoplexy, when associated with the above named heart-disease,

(viz., hypertrophy of the left ventricle,) is found to occur without any recognisable anomaly of the cerebral vessels; and on the other, because bronchial and pulmonary hæmorrhages are frequent in the cardiac diseases referred to. It follows, therefore, that these affections are only in very rare cases to be referred to an increased impulse propagated from the hypertrophied right ventricle, and that in the most numerous cases they are the result of an excessive fulness of the whole vascular apparatus of the lungs, induced by the obstruction opposed to the emptying of the pulmonary veins into the left side of the heart.

These diseases of the heart attack individuals of every age, not even excepting the fœtus, but they occur more frequently in advanced life than in childhood and adolescence, simply because the different causes favorable to their active development have been for a longer time in operation, and the system is no longer equally able to resist disease.

They frequently prove fatal in consequence of the secondary diseases to which they give rise, and often produce sudden death, especially by paralysis of the hypertrophied organ, hyperæmia of the lungs, rapidly developed pulmonary œdema, or cerebral hæmorrhage.

They are further worthy of notice on account of the immunity from tuberculosis, which they ensure to those affected by them; and it may be generally remarked, that the immunity which is yielded by the most various anomalies is always dependent on this class of diseases of the heart.

B. *Abnormal Smallness.*

Anomalous smallness of the heart appears under *two essentially different forms*, being either *congenital and original*, or the result of atrophy—*atrophied*. Abnormal smallness from either of these causes is of incomparably less frequent occurrence than excessive size.

The *former* of these conditions is occasionally associated with a fœtal conformation of the heart, patency of the foramen ovale, and even with more considerable malformations; but many cases present exceptions to this rule. The degree of abnormal smallness varies, the heart of the adult being in

some well marked cases no larger than that of a child of six or seven years of age. This condition appears to be most common in the female sex, and is not unfrequently connected with retarded development of the sexual organs, especially where this arrested development affects the whole system. The opinion expressed by Laennec, that frequent syncope is dependent on a heart too small in relation to the body of the individual, is worthy of observation.

Atrophy of the heart accompanies, to a certain extent, all general wastings of the body, being commonly observed after typhus, and especially in marasmus, in consequence of tuberculous and cancerous secondary formations and their disintegration. An atrophied condition of the heart is also occasionally produced by pressure and want of space, as for instance by bulky secondary products in the mediastinum, and is, moreover, also the result of pericarditis, accumulations of fat on the heart, &c. Contraction of the openings of the coronary arteries is an important and influential cause.

The heart itself differs under these circumstances, the tissue being either tough, and in that case usually of a reddish brown colour, or relaxed, easily torn, of a rusty fawn-colour, and a faded appearance. According to Bouillaud, three different forms may be established in reference to the cavities of the heart, viz.:

a. Simple atrophy, wasting (attenuation) of the walls, with a normal condition of the capacity of the cavities.

b. Eccentric atrophy, attenuation of the walls with dilatation of the cavities.

c. Concentric atrophy, a normal or even an increased thickness of the walls, with contraction of the cavities; this is the most common form.

In the first form the volume of the heart is contracted; in the second it may be contracted, normal, or augmented; in the third it is constantly and generally strikingly contracted. This last form approaches most nearly to original smallness, with which it may even be confounded.

Besides the above named characteristics of the muscular substance of the heart, other signs of atrophy may be mentioned, as, for instance, disappearance of the fat of the heart, serous infiltration of the adipose cellular tissue at the

apex, the base, &c., in consequence of shrivelling of the opaque pericardium and of the milk-spots that may be present; and, lastly, an unusually winding course of the coronary artery.

Morbid attenuation, atrophy of the endocardium and of the valves, will be considered in the sequel.

§ 5. *Anomalies of Consistence.*

We have already acquired some knowledge of several of these anomalies, to which belong :

An *increase of consistence* in the muscular substance of the heart in hypertrophies, which is occasionally very considerable, especially in the right ventricle ;

A *diminution of consistence* in passive dilatations and in some forms of atrophy ;

A peculiar *diminution of the consistence* of the muscular substance of the heart, associated with decoloration, and as the result of pericarditis, and more especially hæmorrhagic, purulent, and tuberculous exudations, which impart to it something of the character of half-boiled meat. (See p. 137.)

Another form of *diminished consistence*, of which we have only spoken cursorily, and which will be considered more fully in a future page, is that which accompanies adiposity of the heart.

The cases that have been regarded by many observers as *softening of the heart's substance*, most probably belong to one or other of these forms of extreme diminution of consistence. It is not unlikely that many of these may have originated in an inflammatory centre in the tissue of the heart.

The diminution of consistence, or a relaxed condition in which the tissues can be easily torn, and which is occasionally observed as the result of typhus, is a mere symptomatic and simple diminution of consistence not depending upon any disturbance of texture.

Softening of the valves will be considered when we speak of the diseases of these structures.

§ 6. *Separations of Continuity.*

To this class belong :

a. *Wounds* of the heart produced by sharp thrusting instruments, as well as by the penetration of fragments of the

ribs, sternum, &c. Such wounds, whether superficial or sufficiently deep to penetrate into the cavities, may injure the heart from different directions and at one or more points.

b. Ruptures of the heart induced by violent shocks implicate, according to circumstances, different portions of the heart, very frequently to a great extent.

c. Spontaneous Ruptures or lacerations of the heart. (*Cardiorhexis, Ruptura cordis spontanea.*) These are the most important of this class of lesions.

Such spontaneous ruptures affect either the walls of a cavity of the heart, a papillary muscle, or its tendinous portion only, a trabecula carnea, or a valve,—the first of these being, however, by far the most common. The left ventricle is the most frequently lacerated, the right only comparatively rarely, and the auricles most rarely of all. When there is laceration of the left ventricle, the lesion almost invariably affects the convex or anterior wall, and generally its middle portion near the septum, the cases being very rare in which the plane or posterior wall is lacerated. The laceration may generally be observed on the external surface of the heart in the form of a fissure, varying in length, and inclining inwards in the direction of the septum. This lesion presents a different appearance in the interior, for here we observe that the muscular substance is, as it were, bruised and crushed over a large extent, near the inner surface, the rent exhibiting either a straight or an oblique course, or in some cases a deep ramified cleft. A coagulum is very commonly found interspersed in the interior among the trabeculæ, or it occasionally fills up the whole cavity.

There is usually only one rent present, but there are occasionally two or even more rents, which are either wholly separate or connected under the surface, and appear at different distances from one another, even in wholly different compartments, as, for instance, simultaneously in the left and the right ventricles.

The investigation of the causes that give rise to these lacerations is a subject of great importance. However much we might be disposed to believe that a heart having thin, relaxed walls, which can be easily torn, would be pre-eminently liable to rupture, or that this lesion would be more common in

those portions of the heart in which the walls are thinnest, such is by no means the case, for laceration takes place, as we have already seen, precisely in the thickest and strongest portions of the heart, and is usually found to occur where the organ is in a hypertrophied condition. On submitting such cases to a more careful investigation, we find, however, as has been already in part shown, that the portion referred to, viz., the left ventricle, in consequence of the diseased condition in which it is often found, especially when combined with hypertrophy, may be so intensely predisposed to spontaneous laceration that this lesion of the heart may occur during a condition of complete bodily and mental repose, and not merely under circumstances of increased action from various exciting causes.

Among the morbid conditions predisposing to rupture, we must place the various fatty conditions of the heart, more especially those which we have mentioned under the head of hypertrophies and dilatations, as a frequent form of textural lesion, in which the muscular substance of the heart assumes a dirty yellowish discoloration, may be easily torn, and becomes loose and flabby. Laceration of the heart is also frequently occasioned by centres of inflammation in the earlier stages, seated in the muscular substance. Contractions of the opening of the aorta may also be included among the remote causes, whilst advanced age affords a specially predisposing cause.

These lacerations, as well as penetrating heart-wounds, generally terminate speedily in death. It has been asked where we are to seek the cause of the speedy occurrence of death in those cases where the quantity of blood extravasated in the cavity of the pericardium is not sufficient to account for the fatal termination of the disease. Some have referred the cause to the implication of the function of the heart itself, in consequence of the extravasation and of the separation of numerous muscular fibres in extensive wounds of this organ. Besides these causes, Bouillaud has advanced an opinion deserving of attention, that death results from syncope,—anæmia of the brain—owing to the sudden abstraction of blood induced by extravasation from the left ventricle.

Penetrating heart-wounds are not, however, invariably rapidly

fatal; life being in some cases considerably prolonged, while it would even appear, in accordance with some observations selected from a large number of cases, that wounds of the heart may be occasionally followed by recovery. The fact that death does not immediately ensue has been explained on the ground of the narrow and oblique course of the wound, and by the different position and crossing of the various wounded layers of the muscular substance. In many cases the wound has been closed by some portion or the whole of the instrument, or even a fragment of a rib, remaining imbedded in it.

It is difficult to answer the question whether the rent occurs in spontaneous ruptures, during the systole or the diastole. Many (Pigeaux, amongst others,) are of opinion, although without sufficient ground, that it generally takes place during the diastole. Judging by analogy with lacerations of the voluntary muscles, it must take place during the systole. In proof, however, of the frequency of its occurrence during the diastole, the fact might be advanced that the course of the rent in the heart, when contracting after the hæmorrhage, is not straight, but angular or zigzag, in consequence of the disturbed position of the different muscular layers.

It is equally difficult to determine, generally or in any individual case, whether the rent has affected the whole thickness of the heart at once, or whether it has proceeded gradually, till it finally penetrated the whole thickness of the wall, and whether it began at the exterior or in the interior. According to our observations on this subject, the rent begins, in most cases, in the inner muscular layers. The laceration of a papillary muscle, or of a trabecula carnea, is of very rare occurrence, and the conditions on which it depends are probably the same as those of fissure of the heart's wall.

Laceration of the tendons of the papillary muscles and of the valves probably always depends on the relaxation and lacerability of the tissue, associated with inflammation of the lining membrane of the heart (endocarditis). It not unfrequently acquires importance by the valvular insufficiency to which it gives rise. *Laesiones continui* of the valves will be more fully considered in a future page.

§ 7. *Diseases of Texture.*

a. *Hyperæmia, Anæmia.*—We are not acquainted with any special condition characteristic of *hyperæmia of the heart*. Occasionally however, hyperæmia, as it manifests itself in the hypertrophies and dilatations which arise especially from stenosis, and in asphyxia in new born infants and adults, is marked by the dark colour of the muscular substance of the heart, and by a fulness of the vessels, more particularly of the veins, and in its more highly developed stages, by slight extravasation in the form of ecchymoses, about the size of millet-seeds or lentils, especially in the external strata and near the base of the heart, at the auricles, and in the vicinity of the origin of the arterial trunks.

Apoplexy of the Heart, manifested by an extravasation of blood into the muscular substance,—a suffusion of the muscular tissue,—is a symptom of no importance in the various degrees of laceration of the heart.

Anæmia of the Heart is probably often overlooked on account of the indistinct signs by which it is characterised. Such a state constitutes, however, a very important (but as it would appear, a hitherto disregarded) morbid condition, as we may learn from the contractions and final obliterations of the openings of the coronary arteries occurring in diseases of the aorta.

b. *Inflammations.*—After having spoken of inflammation of the external investment of the heart—pericarditis,—it still remains for us to notice *inflammation of the lining membrane, and of the muscular substance of the heart.*

1. *Inflammation of the Lining Membrane of the Heart, Endocarditis.*—It is only in modern times, and from the observations of Bouillaud, that this species of inflammation, under the name of endocarditis, has been shown to be the special basis of numerous consecutive heart-diseases. The importance of the subject, both intrinsically and with reference to the different opinions advanced regarding the frequency of the disease, the absence of any well founded data for its correct diagnosis after death, (notwithstanding the many attempts made for their establishment,) and, lastly, our still inaccurate knowledge of its course, its termination, and sequelæ, &c., have determined

us to precede our general notice by a few explanatory observations; and at the same time we would simply remark, as will be seen in this section, that we have arrived, with reference to some points, at a totally different conclusion from the opinions usually expressed regarding endocarditis.

The endocardium corresponds with the inner coat of the vessels, and consists essentially, besides the epithelium, of a longitudinal fibrous coat, (Henle,) under which there is a very considerable layer of elastic and cellular tissue, which is most distinct in the auricles, and especially in their atria, and on which rests the muscular substance of the heart. In the left side of the heart, more especially in the left auricle, a layer similar to that of the circular fibres of the arteries is occasionally found under the longitudinal fibrous coat. This compound investment covers the trabeculæ carneæ, the papillary muscles, and their tendons, while the true endocardium invests the valves also, which, however, can only be regarded as duplications of that membrane, if we consider them as essentially composed of a fibrous tissue supplied with vessels. Besides this fibrous tissue, which is composed of a cellular-fibre-like substance, and delicate nucleated fibres, we also find unstriped muscular fibres in the auriculo-ventricular valves in individuals having a robust and muscular frame. The internal layers of this integument (the epithelium and longitudinal fibrous coat, which constitute the true endocardium) are devoid of vessels; but such is by no means the case with respect to the subjacent cellular tissue, which is permeated with numerous elastic fibres, or with the muscular substance of the heart. The endocardium, as we find in dilatations of the cavities of the heart, and in enlargements of the valves, arising from dilatations of the ostia, is capable of undergoing considerable expansion and attenuation. It is much thicker in the left side, and especially in the left auricle, than in the right side of the heart.

The relation of the true endocardium (the epithelium and the longitudinal fibrous coat) to the subjacent layer furnished with vessels, corresponds with that existing between the inner coat of the veins and their external coats. This condition affords *à priori* evidence of the possibility of inflammation of both coats, considering it, in its usual sense, with

exudation of the free surface, whilst there is no inflammation, properly so called, in the inner coat of the arteries, at least not in the larger vessels, having a thick, yellow, muscular coat of circular fibres. The actual seat of these inflammations is the cellular substance lying under the endocardium and the inner coat of the vessel; we must, therefore, suppose, that in cases where products of inflammation are deposited on the inner surface of the heart or of the vein, the exudation must have penetrated through the permeable texture of the endocardium or through the inner coat of the vessel, or that the latter has been removed, either by solution or fusion, by means of the process of exudation. The latter condition will naturally be found to be of most common occurrence in inflammations having a purulent ichorous exudation. Such alterations manifest themselves by opacity, lacerability, and a felt-like porosity of the endocardium, excoriation of the subjacent layers, &c.

We learn from the foregoing observations how far the designation *endocarditis* is applicable to inflammation of the lining membrane of the heart. Thus, for instance, it is evident that since the endocardium, like the inner coat of the vessels, is non-vascular, it cannot be the seat of inflammation, which affects merely the tissue lying immediately below it, which is furnished with vessels. We purpose retaining the term *endocarditis* in this sense, discarding its use in reference to the valves, for which we shall simply retain the designation of inflammation of the valves.

Although *endocarditis* is a disease of very frequent occurrence, it must not be supposed that the term is applicable to all the diseases ascribed to it, its products, and sequelæ, for, as we shall soon learn, many morbid conditions of the valves of the left side of the heart, especially of those of the aorta, are the products of the same process which manifests itself in the arteries as a morbid deposit on the inner coat.

Endocarditis attacks different portions of the lining membrane of the heart, affecting in some cases the endocardium covering the inner surface of a cavity, the papillary muscles, and the trabeculæ, in others that of the valves, while in others again it affects both. *Endocarditis* of the valves is the most

frequent and the most important from the consecutive heart-diseases to which it gives rise. We will consider the signs of both under one head, referring specially to the peculiar characters of endocarditis of the valves.

1. *Redness and Injection.*—In order that these conditions may be regarded as the manifestation of inflammation, it is necessary that the former should be the result of the latter (inflammatory injection or stasis), or that it should depend on an exudation containing hæmatin into the tissue. This latter form of redness is always found together with other signs of inflammation, and usually presents a mottled appearance. Where the redness cannot be referred, at least in part, to the above cause, it cannot be regarded as a sign of inflammation. Now, in point of fact, all the different forms of redness of the endocardium, which have been generally described as characteristic of endocarditis, belong to the latter class, and the descriptions given of these various forms evidently show that they are mere modifications of that redness which depends on infiltration of the tissue with hæmatin. There are, however, so few opportunities of detecting the peculiar redness of the endocardium arising from injection, as our own numerous observations can testify, that it would not be surprising if anatomists, instead of committing an error of this nature, had wholly denied the existence of inflammatory redness in endocarditis. It is only in the first stage of the disease that a true redness and injection can be observed through the endocardium; it is only, therefore, in the very rare cases in which death occurs in the earliest stage, either from this or some other disease, that this condition of redness can be perceived. In most cases a redness from imbibition, resulting from the diseased condition of the blood, is *actually* present, and renders it extremely difficult and almost impossible to discover the redness from injection, which differs wholly from the above named red colorations, and is constantly of a pale, rose-red colour, whose tint is subdued by the endocardium covering it. Its appearance is never that of a saturation of the tissue, and its stripe-like, ramifying course, corresponding to that of the vessels, may the more easily escape detection, when it is concealed by the presence of a simultaneous red coloration, arising

from infiltration. In most cases we are unable to perceive this redness from the circumstance of its being wholly masked by the conditions we are about to consider, viz. :

2. *Opacity and Thickening of the Endocardium.*—In consequence of the extension of the process, the endocardium at various differently-sized spots is rendered opaque, whitish, and milky, whilst at the same time it becomes more or less thickened and swollen. This opacity and thickening depend on the deposition of the product of inflammation in the tissue of the endocardium and the subjacent stratum, where it either solidifies or exerts a relaxing, macerating, solvent action on that tissue. The opaque and thickened parts are not clearly defined, but appear gradually to lose themselves in the adjacent portions of the endocardium. Valves affected by endocarditis exhibit a remarkable degree of thickening, because the substratum of infiltration—the tissue occurring between the two lamellæ of the endocardium—is here accumulated in larger quantity. The shining smooth appearance of the endocardium vanishes with the increase of the opacity and thickening, and it then acquires a *dull*, velvet or felt-like and *rough* surface.

3. The whole of the lining membrane of the heart acquires a *looseness of texture*, and then readily admits of being torn, while the true endocardium is easily detached. In inflammation of the valves, their fibrous tissue very frequently appears to be in an extreme state of looseness and relaxation.

4. *Products of Inflammation.*—To this class belongs the above named infiltration of the endocardium and of the subjacent tissue, but the question here arises, whether there is also exudation on the free surface of the endocardium, and how far such a condition is necessary to establish the existence of endocarditis.

The fact of such an exudation being deposited on the free surface of the endocardium in most cases of endocarditis, is rendered highly probable, not only from the results of pathological investigations, and the analogy presented by inflammations of other similar structures, especially the serous membranes, but still more so from the symptoms manifested during life. This exudation at the moment of its production merges into, and is taken up by the mass of the blood, where, in accordance with its character and intensity, it gives rise to the

different general symptoms manifested during life, and to the characteristic secondary processes observed in the capillary system in endocarditis. In many cases, however, this exudation, doubtless in consequence of a very high degree of coagulability, remains on the inner surface of the endocardium in the form of a membranous coagulum, having a delicate felt-like, or shaggy free surface, which we have rarely an opportunity of seeing in its original condition, but which may very frequently be subsequently observed under different forms, but most distinctly in the form of milk-spots on the endocardium. In endocarditis of the valves it commonly manifests itself in the form of felt-like or granular masses, under which the valve appears rough, loose in its texture, and excoriated, and it then, in part, constitutes the so-called vegetations of the valves of the heart. We shall subsequently speak of a purulent exudation on the endocardium.

5. *The so-called Vegetations or Fibrinous Coagula* which occur under the most various forms, more especially when they appear on the valves of the heart, are generally, and without exception, regarded as characteristics of endocarditis. As, however, they are not invariably direct products of an exudative process, but, on the contrary, in some cases wholly, but more frequently only in part, indirect effects of endocarditis, since they undoubtedly also appear independently of that disease, we cannot regard them as signs of endocarditis without some limitation in accordance with what we have already stated, and with that which we purpose advancing in a subsequent part of this work, when we proceed to treat specially of vegetations in the heart.

It follows from the above considerations that the anatomical characteristics of endocarditis are very inconsiderable in number, when compared with those of other inflammatory affections; redness and injection are only seldom to be observed, an inflammatory product on the free surface of the endocardium is not always to be detected, and the vegetations are only conditionally a sign of endocarditis. There remain, therefore, as the only constant signs, opacity and thickening of the endocardium, with the disappearance of the smoothness and polish of its surface. But as these conditions of opacity and thickening of the endocardium may, as we have already remarked, be

produced by a process wholly different from that of endocarditis, it will be readily understood how difficult is its diagnosis, and how easily its products may be confounded with those of some wholly different process.

In the above delineation we have purposely limited ourselves to the most important points, in order to give a general sketch of the endocarditic process; and with the further view of not disturbing our readers by any superficial details, we have described only the characters presented in the most numerous and common cases of endocarditis. We purpose considering this subject with the completeness which its importance demands, and we will then treat of all those points that have been neglected in the present portion of our work. The following observations will contain a notice of many of the more uncommon events occurring in the course of endocarditis, and of many appearances and processes which have merely been briefly indicated in the preceding delineation, together with the terminations, sequelæ, &c., of the disease.

a. In intense forms of endocarditis, a *separation of continuity* of the structure affected by the inflammation not unfrequently manifests itself as a highly important occurrence. It may occur in different ways, either as laceration of a valve, or of one or more of the tendons of the papillary muscles, or of the endocardium on the wall of the heart. This separation of continuity is the final result of a maximum degree of inflammatory loosening of the tissue. The margins of the fissure are generally jagged, and serve as the places of deposit for a large number of vegetations. The tissue of the torn structure, as for instance of a tendon, is usually considerably reddened, infiltrated by inflammatory products, and easily torn. Laceration at the wall may give rise to the formation of aneurism of the heart, whilst, if it affect the valve, it may, under certain circumstances, occasion valvular aneurism.

b. *Endocarditis with purulent exudation* is not of very uncommon occurrence; and although the recognition of the seat and position of pure pus, as a free product, is, in most cases, impracticable, it is not difficult to prove the extreme probability of the existence of such a process. The loosening of the tissue, the want of polish, and the felt-like character of the endocardium, are very strongly marked in the centre of inflam-

mation, and hence these lacerations frequently occur. In these cases a purulent product mixed with blood is generally found infiltrated into the tissue, if not at the surface of the endocardium, whilst abscesses are occasionally found to have spread themselves over a various extent of surface below the endocardium, in the cellular and adjoining muscular strata, deep in the tendons, and in the tissue of the valves. Finally, the process of suppuration being established, an ulcerous separation of continuity will be effected in various ways, in the endocardium of the walls of the heart, in a tendon, or in a valve. The vegetations deposited on the ulcerated surface and its margins are remarkable for their excessive number, their inconsiderable consistence, bad colour, and their tendency to purulent disintegration. The secondary processes in the capillary system terminate in purulent solution, whilst the intensity and malignant character of the general symptoms during life lead us to conjecture that some deleterious substance has been taken up into the blood.

c. Endocarditis is probably always an acute disease; it may, however, frequently recur, and at the same spots; but we cannot admit the existence of a chronic form of the disease, unless, according to Bouillaud's incorrect view, we regard as such the symptoms manifested during life by its products, and the further development and metamorphosis of those products, that is to say, the terminations and sequelæ of endocarditis as given below.

1. *Exudations on the free surface of the Endocardium* in the form of agglutinated, whitish, or bluish-white laminae of different size and form, resembling in appearance a serous or fibro-serous membrane, under which the endocardium appears normal, or scarcely at all opaque. They at one time appear in the form of narrow stripes, at another in that of more considerable, irregular plaques or patches, varying from the size of a silver groschen to that of a zwanzigerstück, [a coin rather larger than a shilling], and admitting of being easily removed from the endocardium, over which they are in general smoothly drawn or occasionally compressed together in folds. They are most frequently observed in the left side of the heart, at the upper part of the septum towards the aortic opening, where they are puckered and drawn aside into plaits by the

blood flowing over them. Their texture resembles that of the longitudinal fibrous coat, and they consist in some cases of thick stiff fibres, and in others of soft fibres of areolar tissue. The facility with which the agglutination of the inner milk-spots are severed, and the laceration of texture occasioned by their separation, cause them to differ very widely from other structures. The milk-spots are, however, almost always sharply defined in these cases.

2. *Permanent Thickening of the Endocardium and of the Subjacent Tissue* becomes the more considerable in proportion to the intensity of the endocarditis, and the frequency of its occurrence at the same spot. It is generally occasioned either by infiltration into the tissue, or by exudation that has solidified and become organised on the free surface of the endocardium; the former of these exerts, however, a preponderating influence, as is especially observed in the valves. Thickening is manifested in the walls of the heart in the form of patches of various extent, in some of the trabeculæ as a tendinous ring or sheath, in the papillary muscles as a tendinous covering over their extremities, in the tendons themselves as a wad-like or spindle-shaped thickening, and in the valves as a more or less uniform thickening of their free margins, extending from thence to various distances, and even across the valve towards their margin of insertion. The diseased tissue appears opaque, thick, tough, and of a white colour, inclining to yellow; and it is with difficulty that the free exudation and the tissue infiltrated by solidified products of inflammation, which constitute the principal elements of the morbid mass, can be torn or split asunder, both having coalesced, and presenting a single fibroid and compressed texture.

The thickening of the tissue of the wall of the heart is often made more apparent in endocarditis by the association of inflammation of the contiguous stratum of muscle to various depths, which gradually passes into induration, and leaves a fibroid callus in the place of the muscular fibres.

3. *Coalescence* is frequently associated with this thickening of the tissue. As the thickened tissues coalesce with the free exudation, so also the latter may occasion a fusion of various tissues. In this manner the trabeculæ enclosed in tendinous sheaths unite with one another or with the walls of the heart, while the same process may be observed amongst the separate

points of a papillary muscle, or the tendons of a papillary muscle may merge into either one or several strings, or the different valves may coalesce with one another, or with the wall of the heart or of the vessel.

4. This fibroid mass of exudation exhibits here, as in other places, a marked tendency to shrivel, by which means a *shrivelling or shortening* of the thickened structure takes place. To this class belong shortening of the papillary tendons, and a shrivelling of the valves associated with various malformations. The wall of the heart is either very indistinctly or not at all shrivelled, since it is raised by the substratum of muscle in those cases where the latter has retained its normal texture and function; the adventitious product is expanded rather than shrunk, owing to the great influx of blood in those cases in which the muscular substance of the heart has been reduced to a state of paralysis by the action of inflammation, or has suffered a change of texture.

5. *Calcareous Concretions* become developed sooner or later in the fibroid secondary product, and appear in rare cases in the form of nodular uneven laminæ in the thickened endocardium of the wall of the heart, and more frequently as simple nodular or ramified strings or rows, or even as amorphous masses of various thickness in the tissue of the thickened valves, and of the thickened papillary tendons which are generally fused and blended together.

6. We have already fully considered the subject of endocarditis terminating in *Suppuration*.

d. Although endocarditis is generally characterised by the terminations and sequelæ already indicated, the cases in which it terminates by a perfect cure are not of very rare occurrence, as we learn from careful observations on the living subject, and by a correct interpretation of the appearances presented after death. This favorable termination depends occasionally on a complete resolution; or, in other words, on the absorption of the products of inflammation deposited in the tissue, and on the fact that the portion of the free exudation which is solidified on the endocardium, and the vegetations that may be present, are gradually taken up by and mixed with the mass of the blood in the form of finely divided molecules. In some cases fragments, from their size and position, do not constitute

an impediment to the circulation, may remain; or, again, in other cases partial thickening of the valves is counteracted and rendered innocuous by their becoming attenuated at one or more points, by a shortening of one or more of the papillary tendons, or an elongation of the muscle or of the extremity of the diseased valve.

e. The Vegetations on the Valves above referred to undergo different metamorphoses, as we have already seen, and as will be made more apparent in the sequel. We will here specially notice:

1. *Their Gradual Diminution and Final Disappearance.*—According to numerous highly interesting analogies, it would appear that these conditions depend on an actual waste of the fibrinous coagulum. They undoubtedly occur very commonly, and from a comparison of the frequent or almost invariable appearance of very numerous and extensive vegetations on the valves in recent endocarditis, and their insignificant character and occasional absence in obsolete cases, it appears evident that, in the course of time, they become considerably diminished, and at length entirely disappear.

2. *Ossification and Calcification* of these vegetations are metamorphoses, which, although of frequent occurrence, have not hitherto been duly considered. They constitute a special form of valvular ossification, which has never yet been duly considered.

f. The secondary Coagulation of Blood in the capillary system, together with its metamorphosis, which presents a highly important indication of the endocarditic process,¹ has also been disregarded by observers. It indicates the most important phenomenon manifested during endocarditis, namely, the formation of a product on the free surface of the endocardium and its absorption into the mass of the blood, and consequently shows the equal importance of endocarditis and inflammation of the vessels, (namely, of the veins,) while it moreover tends to elucidate the symptoms of disease during life. It is more constant than in phlebitis, inasmuch as, from the absorption of the inflammatory product, no coagulum can be formed in the heart of a similar nature to those which occur in the veins, and hence there can be no immunity afforded against a poison-

¹ Oesterr. med. Jahrbücher, B. xix, St. 3.

ing of the whole mass of the blood. This process probably, on this account, constitutes an important means of diagnosing between obsolete endocarditis and a form of hypertrophy of the endocardium, and more especially of the valves, which is induced by depositions from the blood; but this subject we will presently consider more at large.

It is of common occurrence in the spleen and kidneys, but is seldom found in the lungs, excepting in the very rare cases of endocarditis of the right side of the heart. The secondary processes which result from endocarditis depositing a purulent exudation, and terminating in suppuration, are less limited to these organs of hæmatisis, and manifest themselves as metastases in the subcutaneous cellular tissue, in the mucous membranes, &c.

The process commonly called Phlebitis (but more appropriately termed Angioitis capillaris) consists, as far as we know, like that observed in a larger vessel, (namely, a vein,) in a coagulation of the blood in the capillaries, and a metamorphosis of the coagulated fibrin, varying in accordance with the quality of the absorbed product. Since endocarditis, in ordinary cases, yields no deleterious product (pus or ichor), the metamorphosis consists in a conversion of the fibrinous coagulum into a fibroid mass, with obliteration of the vessels, and so great a degree of obsolescence of the affected tissue of the diseased organ, that the whole resembles a cellulo-fibrous callus, which shrivels to a callous, whitish or black cicatrix, containing pigment. It is not improbable that the process may terminate in resolution, or, in other words, in the solution of the coagulum, and thus leave no trace of its existence. In the very rare cases in which endocarditis deposits a purulent product, the coagulum in the capillaries becomes decomposed into a fluid, which is more or less purulent, according to its elementary composition, while there is consecutive fusion of the walls of the vessels and the diseased tissue, the result of which is the formation of an abscess, or so-called purulent metastasis. This form of endocarditis may result in a true process of exudation in the serous and synovial membranes, and even in the parenchyma, in consequence of the diseased condition of the blood induced by the morbid product.

g. Endocarditis by its proximate, no less than its secondary

results, and therefore by a twofold local cause, may give rise to *Dilatations of the Heart*. As we have already observed in treating of these diseases, the form of dilatation thus occasioned is of a passive character, and depends on paralysis of the muscular substance of the heart, which is implicated in the inflammation. The dilatation is moreover mechanically increased in the more remote sequelæ of extensive endocarditis by the continuance of a morbid condition of the valves, which are almost invariably implicated; and in these cases a moderate degree of hypertrophy is gradually associated with the dilatation. The dilatation must also, as is evident, be more considerable from its very origin, and must be of a more decided passive character, where endocarditis has been combined with pericarditis and carditis; and where the latter affection is of a very intense and deep-seated character, endocarditis may give rise to true aneurism of the heart.

Endocarditis occurs with a preponderating degree of frequency in the left side of the heart, where it is also generally present in the very rare cases in which it attacks the right side. In the case of the former, both the ventricle and the auricle are affected, while in the case of the right side of the heart, the ventricle is the special seat of the disease. The auriculo-ventricular valves of the left side are more frequently diseased than those of the right, whilst many morbid conditions of the aortic valves cannot be actually referred to an endocarditic origin.

An interesting exception to these relations is presented in the fœtus, where endocarditis is much more frequent in the right side; and many of the cases of contraction of the openings of the right side, which are met with in childhood and youth, are undoubtedly congenital and of fœtal origin. There are, moreover, many anomalies of the arterial opening, especially of the right side of the heart, and of its valves, which are commonly regarded as malformations, (as, for instance, contraction and occlusion of this opening, and an abnormal condition of the trunk of the pulmonary artery,) which are most probably the results of endocarditis already existing in an early period of fœtal life, and which give rise to many arrests of structure within the heart. We may undoubtedly explain in a similar manner the many endocarditic metamorphoses observed in the hearts of persons suffering from cyanosis.

From what has been already stated, it will appear, that endocarditis occurs in the foetal condition as well as after birth. Youth and adolescence are the periods in which this affection is most frequently manifested.

The most important diseases with which it is associated, are its primary combination with pericarditis, and, whether this be present or not, with inflammations of serous membranes, namely, those of the synovial membranes—rheumatic inflammations of the joints. Valvular endocarditis, implicating the tendinous insertion of the mitral valve, when combined with pericarditis, is extremely important, owing to the peculiar formation of the consecutive metamorphoses. Thus, for instance, we observe that the calcareous band developed at the tumefied point of insertion, not unfrequently expands into an osseous mass seated in the pseudo-membranous agglutinating medium between the pericardium and the heart. Endocarditis is also occasionally combined with carditis—inflammation of the muscular substance,—and this combination is then the common occasion inducing aneurism of the heart; while in some cases endocarditis may be merely an incidental combination, arising from some centre of inflammation in the muscular substance adjoining the endocardium.

To this class belong the combinations with croupous pneumonia, acute inflammations of the periosteum, acute otitis, &c.

Endocarditis and its sequelæ are not unfrequently met with in combination with Bright's disease, which is probably to be explained by the fact that this heart-disease becomes associated with disease of the kidneys in consequence of the abnormal condition of the blood.

Many of the anomalies already partially considered occur as the remote and indirect sequelæ of endocarditis. Foetal endocarditis, at an early period, obstructs the completion of the inner structure of the heart, by means of the results to which it gives rise, and especially by contracting the openings of the heart; when it occurs at a later period and after birth, it obstructs the involution (closure) of the foetal passages. In subsequent periods of extra-uterine existence, many of the diseases of the different systems and organs considered under the head of dilatation and hypertrophy, may still be traced to the dilatation of the cavities of the heart and the anomalies of the

valves, (contraction and insufficiency,) which have their origin in foetal endocarditis.

Hypertrophy and Atrophy of the Endocardium.—By these conditions we purpose indicating a thickening of the true endocardium, (which, in respect to the main character of its composition, corresponds to the inner coat of the vessels,) by a morbid deposition from the blood of a substance which becomes metamorphosed into the layers of epithelium and longitudinal fibres composing the endocardium. This excessive deposition of new layers of the endocardium is a process which occurs in its most fully developed form in the arteries, and more especially in their main trunks, and will be duly considered in the appropriate place. Its proximate result is a thickening of the endocardium.

This morbid condition acquires additional importance from the facility with which it may be, and no doubt very frequently is, confounded with endocarditis and its products, which it greatly resembles, and with which it is often found associated. We have, on this account, thought it best to devote the closing part of the present section on endocarditis, to the consideration of this subject, however unusual such an order of arrangement may appear. (See p. 153.) The following remarks on the peculiar characteristics of this affection will clearly exhibit the differences which distinguish it from the endocarditic process and its products.

In the lower degrees it is only by a careful investigation that we can discover any undue thickening of the endocardium. The colour of the muscular substance is less clearly discernible, while more strata than usual must be removed before we reach the layer of cellular tissue interspersed with elastic fibres, which is situated under the endocardium; moreover we clearly observe that the innermost layers are lighter and softer, and that the tissue which constitutes the longitudinal fibrous coat is less developed and more moist.

In this manner new depositions of layers of endocardium, either with or without an epithelial investment, are frequently found to cover one or more of the cavities of the heart, (the ventricle or auricle of the left or both sides,) together with the corresponding valves.

When this process of deposition has been frequently repeated,

and the thickening of the lining membrane of the heart is correspondingly increased, this condition will be easily recognised. In these higher degrees of intensity we very frequently observe, as in the arteries, that the endocardium exhibits, at more or less well defined spots, portions of thicker surface in the form of islands or patches, while we at the same time remark that the valves, more especially those of the aorta, have been considerably thickened and enlarged by the deposit.

The opalescent translucence and stratification of the deposit and the uniform texture of the combined lamellæ, distinguish it from the products of endocarditis, from the loosely attached, bluish white, opaque milk-spots, and from the fibroid thickening of the endocardium, which cannot, without extreme difficulty and effort, be separated into strata, and which exhibits greater density and dryness of its tissue, and evidently consists of fibrous or areolar tissue. The absence of redness and injection in every stage and of vegetations and secondary processes (metastases) in the capillary system, distinguishes it from the endocarditic process. The existence of the process of deposition in the trunk of the aorta affords us further diagnostic aid in determining hypertrophy of the endocardium.

A correct diagnosis, which has for its object to determine both processes generally, and to distinguish the special share taken by each in the anomalies under consideration, is rendered more difficult in those cases in which, as we have already observed, the products of endocarditis occur simultaneously with the condition we term "excess of endocardiac formation," and which is indeed very commonly favoured or even occasioned by the residua of endocarditis. A peculiar difficulty presents itself, when the deeper or older deposits lose their transparency, and become completely opaque, white or faded, in consequence of an atheromatous process, or of a metamorphosis tending to ossification. They may be distinguished from endocarditic products on a closer inspection, by the occurrence of a large quantity of molecules, consisting of albumen, fat, and calcareous salts, deposited in the different strata.

This metamorphosis never, so far as we know, proceeds on the walls of the heart, beyond the incipient stage above indicated; we have never found it developed into the true atheromatous process, nor have we ever been able to ascertain that this pro-

cess formed the basis of any of the numerous cases of aneurism of the heart which we have examined. The valves, however, occasionally present the appearance of an incipient atheromatous disintegration of the deposit, while ossification of the deposit on the valves, more especially on those of the aorta, is very frequently a final result. It is highly probable, moreover, that all the forms of ossification of the valves which become developed in the advanced periods of life, belong to this class.

Hypertrophy of the endocardium is limited almost exclusively to the left side of the heart, and of the two arterial trunks it only attacks the aorta. The aortic valves and the left ventricle are more frequently and more intensely affected than the auriculo-ventricular valves and the auricle. The aorta is at the same time diseased in like manner, but generally in a very proponderating degree. The endocardium of the left auricle is, however, excessively thickened in some few cases where there is contraction of the mitral valve.

Endocarditic hypertrophy, like that of the aorta and its ramifications, especially occurs in advanced periods of life, and undoubtedly constitutes the source from whence arise a great number of those diseases of the aortic valves,—as, for instance, thickening, shrivelling, ossification, and insufficiency,—which are slowly developed in maturity and old age, without the pre-existence of endocarditis. This fact presents many points of great interest, when considered in relation to diseases of the mitral valves, which usually occur in young persons as a result of well-marked endocarditis.

This affection is frequently occasioned and favoured by pre-existing dilatation of the heart and contraction of the openings, in consequence of which the blood is detained in the different cavities, and its further circulation impeded.

Atrophy or Attenuation of the Lining Membrane of the Heart is very seldom sufficiently manifested to come under notice. This membrane is certainly found to be uncommonly thin and transparent in some cases of dilatation of the heart; while we have remarked the same appearance in excessively fat hearts.

2. *Inflammations of the Muscular Substance of the Heart, Carditis (in the strict sense of the word), Myocarditis.*—Although inflammation of the *Muscular Substance of the Heart* is less frequent than endocarditis, it is much more frequent

than is usually supposed. Its anatomical characters and its terminations are the same as those exhibited in inflammation of the muscular substance generally, but there are, nevertheless, many points connected with this subject which demand special notice, both on account of their importance and peculiarity.

It occurs independently in the middle layers of the muscular substance most remote from the pericardium on the one hand and from the endocardium on the other, and in original or consecutive combination with pericarditis and endocarditis. The pericardium and the endocardium are always implicated in inflammation of the adjacent layer of muscle and conversely intense pericarditis, and more especially intense endocarditis influence the adjacent structure to various depths. It moreover most frequently affects the true fleshy walls of the heart, but sometimes its trabeculæ, and in some cases both simultaneously.

It also commonly occurs in the form of larger or smaller centres which are in some cases spread over a large portion of one cavity of the heart, (as, for instance, the left ventricle,) in which case, the wall of the heart is found to be affected throughout more or less of its thickness, when the disease is associated either with pericarditis or endocarditis singly or with both conjointly. In some rare cases one portion of the heart is found to be so thoroughly affected, that there are only a few layers of the muscular wall which are not implicated.

The seat of the affection is almost exclusively the left ventricle, which it attacks at every point, although less frequently at the septum; the apex is commonly attacked when the disease is very extensive. The right ventricle is very rarely affected, although we have observed the disease in an intense degree of development in the anterior wall of the conus arteriosus. It is of very rare occurrence, as far as we know, in the auricles. (See our remarks, in a future page, on Aneurism of the Heart.)

Inflammation of the substance of the heart always gives rise to dilatation of the cavity implicated, and this dilatation is proportional to the extent of the inflammation and to the number of its centres. When combined in an early stage with endocarditis, it occasionally results in the formation of an *acute*

aneurism of the heart (of which we shall subsequently speak), in consequence of a laceration of the tissue which has been loosened by the process of inflammation. Finally, as we have already remarked, centres of inflammation are not unfrequently the cause of spontaneous ruptures of the heart.

This affection commonly results in *induration and in suppuration*, although it much more frequently assumes the former than the latter mode of termination.

In the *former* we find, in place of the muscular substance, a white fibroid (cellulo-fibrous) tissue, either in the form of small stripes, or spread over a more extended surface, according to the size of the centres of inflammation and the mass of the inflammatory product; or we may observe, where the indurated product of inflammation is accumulated in larger quantities at definite points and forms a tissue of this nature, nodular, roundish or irregularly shaped, ramified tumours, having the toughness of callus, which protrude either externally, or internally into the cavity of the heart. This form of striped indurations is frequently found to be deposited in the same subject in great quantity on the most different strata of the muscular substance of the heart, especially where an accurate investigation shows us the residua of pre-existing endocarditis, combined with consecutive dilatations and hypertrophy. Professor Bochdalek has drawn attention to this fact and to the frequency of carditis, which has hitherto been overlooked and generally denied.

The more widely extended inflammations of the muscular substance of the heart exhibiting this termination are of especial importance. They affect either the inner layers of the walls of the heart, together with the trabeculæ and the base of the papillary muscles, including the endocardium; or the external layers, together with the pericardium; or, lastly, the wall of the heart throughout its whole thickness, including both the pericardium and the endocardium. Occasionally we find that contiguous portions of the innermost, the middle, and the external layers of the muscular substance of the heart, are in turn attacked. The muscular substance is here found to be replaced by a fibroid tissue, while the walls of the heart, the trabeculæ, and papillary muscles, appear to be converted into a white callous tissue;—a process in which the endocardium so far participates, that it not only enters to a corresponding

extent into the same metamorphosis, and becomes identified with this tissue; but it even generally exhibits a gradually decreasing fibroid thickening beyond the limits of the metamorphosis in the muscular substance. We also observe at the pericardium exudations, which are either well defined, or spread over the whole heart, and have been converted into cellular or fibroid tissues; and these give rise to adhesions.

These generally diffused metamorphoses which affect the wall of the heart throughout its whole thickness, not only exert an influence, in a general sense, on the increase of the dilatation of the respective cavities of the heart by means of the inflammatory process, but also specially on the origin of defined saccular dilatations—*true chronic aneurism of the heart*—which we shall subsequently consider more at large.

The fibroid tissue in the wall of the heart, in the trabeculae, and in the papillary muscles, becomes, not unfrequently, in the course of time, the seat of calcareous deposit, constituting what is termed *ossification of the walls of the heart*, which invariably depends on the pre-existing alterations of texture of the muscular substance of the heart, which we just described.

The termination of carditis in *Suppuration*, which is much less frequent, gives rise to *Abscess of the Heart*.

In accordance with what has been already stated, abscess of the heart is almost entirely confined to the wall of the left ventricle, where one or more accumulations of pus may be present.

They are generally of inconsiderable *size*, being about equal in circumference to a pea, a bean, or a hazel-nut. A more considerable size, if it does not consist in an extension of surface, is indeed incompatible with the continued existence of a recent abscess, since it would speedily be associated with a rupture of the walls.

These abscesses are usually of an irregular *form*, exhibiting various sinuosities, running in different directions.

The muscular substance of the heart immediately adjoining them, is in a condition of purulent infiltration and disintegration; at a somewhat greater distance, it is pale, permeated by a serous or sero-purulent exudation, soft, and admits of being easily torn; while still further from the abscess it is livid, and not unfrequently interspersed with varicose vessels; it is also relaxed.

Under the last named conditions, the abscess may be encysted, in which case it may exist for a longer period, while its contents may moreover become either in part absorbed, or in part condensed and cretified, and the abscess may in consequence be obliterated. Its usual termination, however, where paralysis of the heart does not supervene, will be *its opening* either internally or externally, and, in consequence, or independently of these causes, there will be complete *perforation* of the wall of the heart from laceration of the strata of the muscular substance, which are incapable of further resistance. It frequently happens in internal openings, that the endocardium not only suppurates, but is torn to an extent corresponding with the size of the abscess. Such an opening is followed by a discharge of pus into the cavity of the heart and its absorption into the blood; and very commonly, even before the symptoms of pyæmia have been fully developed, by a swelling of the muscular substance of the heart, owing to the penetration of blood into the cavity of the abscess, and by laceration of the remaining external layers of muscle, that is to say, by perforation.

Some very rare instances of superficial abscesses opening internally may be unattended by perforation, in which case the cavity of the abscess will constitute an acute form of aneurism of the heart, till the pyæmia induced by the discharge of pus into the cavity of the heart ultimately proves fatal. We are not acquainted with any well attested case in which the discharge of pus has been restrained by the mass of the blood flowing into the opened cavity of the abscess, and by the deposition of coagula, or where aneurism of the heart had, in this way, become established for any length of time.

in preceding remarks on endocarditis and carditis, and in the various processes, we have frequently alluded to the importance of this secondary condition. The importance of this secondary condition, however, that we should treat the condition, and we, therefore, purpose devoting the following consideration.

rt, known also as partial, (*Aneurisme du*
g to Breschet, as *consecutive aneurism*,

is a circumscribed dilatation of one of the cavities of the heart, depending specially on a diseased condition of the texture of the endocardium and of the muscular substance of the heart. We retain the designation of aneurism, with its inappropriate accompaniments of "*partial*" and "*false*," because the terms have been universally adopted, and because this condition exhibits in its pathological relations a certain resemblance to that which we designate *Aneurism of the Arteries*. We would, however, at once definitively explain, that we do not consider that there exists any close affinity between these two conditions. In fact, according to our views, this resemblance depends mainly on the circumstance that both conditions are based on an alteration of texture; we will, however, leave it to our readers to compare the two, and to analyse for themselves the special similarities and differences they may be found to present. We are utterly unable to concur in Thurnam's views on aneurism of the heart; nor can we adopt, as the sequel will show, the classification by which he divides aneurism of the heart into numerous species, corresponding to the different forms of aneurism of the arteries.

At the present day we are acquainted with only two essentially differing species of aneurism of the heart, one of which represents an *acute*, and the other a *chronic form*; the former corresponding generally to *false*, and the latter to *true aneurism* of the arteries. We are led, from the numerous observations we have ourselves made, either wholly to discard all other forms, or at any rate to regard those as doubtful which are based on the unsatisfactory researches of other inquirers.

1. One, and certainly a rarer form of aneurism of the heart, is a proximate result of a recent inflammatory process of the endocardium, and probably, also, in great measure of the contiguous muscular substance of the heart, and depends on a laceration of the diseased tissue, which is itself the immediate consequence of its inflammatory relaxation. The blood rushes violently through the rent, which is either limited to the endocardium, or involves with it a portion of the adjacent layers of the muscular substance, and thus disturbs the still uninjured muscular tissue of the heart to various depths. A cavity is thus formed, whose walls consist of the upheaved, lacerated muscular substance, and which is surrounded at its mouth by

a torn and fringed margin of endocardium. The blood poured into this cavity deposits its fibrin in the form of soft coagula, infiltrating the lacerated muscular substance, and occurring on the fringed membranous margin in the different forms of vegetations observed in the valves. This aneurism is developed in an *acute manner*, as may be seen from what has been already stated, and is accompanied by the appearances of recent endocarditis. We have never seen a case in which the walls of an aneurism of this nature had become consolidated into a fibroid, callous tissue; for, in all the cases we have examined, the aneurismal formation was only of recent date, having existed only for a very inconsiderable period after the endocarditis, during the continuance of which it had originated. None of the cases in which an aneurism with solid, callous walls, existed for any length of time after the endocarditis, afford the slightest evidence that it had originated in this acute manner from laceration. The investigations of foreign observers have so far influenced pathologists, that they have begun their inquiries regarding aneurism of the heart with callous walls with the preconceived opinion that a *lesio continui* occurs in the endocardium, as in the so-called *mixed aneurism* (*A. spurium* of Scarpa,) of the arteries; and the difficulties attending the investigation of this form of aneurism of the heart, have greatly contributed to the maintenance of this error, notwithstanding the numerous proofs we have advanced to the contrary.

2. The second form of aneurism of the heart is either the remote consequence of the combined inflammation of the endocardium and of a somewhat thick layer of the muscular substance, or more frequently of inflammation of the wall of the heart throughout the whole of its thickness, accompanied with endocarditis and pericarditis. The inflammation of the muscular substance, by its tendency to induration, promotes the development of a white fibroid tissue, which occupies the place of the muscular fibre in the trabeculæ, as well as in the actual muscular substance of the heart, and coalesces, as it were, on its inner surface, with the endocardium, which is thickened into a similar tissue, and towards the exterior, with cellular or fibroid formations,—the products of endocarditis and pericarditis. This tissue, with its inherent tendency to shrivelling, is unable to resist the pressure and flow of the current of the blood, and by

its yielding and expanding gives rise to circumscribed dilatation of the cavity of the heart. The limits of this dilatation generally correspond with those of the metamorphosis of the muscular substance, extending as far as the point where the muscular fibre has remained undestroyed throughout the whole, or a considerable depth, of the thickness of the wall of the heart. The course of the development of this aneurism of the heart is therefore *chronic*, when considered as a remote result of the above named combined inflammations.

It follows from the above observations, that this form of aneurism of the heart is a *circumscribed dilatation* of one of the cavities, whose walls consist wholly or for the most part of a *fibroid* (tendinous, ligamentous, cellulo-fibrous, callous) *tissue*. This circumscribed dilatation exhibits a *shallow sinus* in the muscular wall of the heart, or an ordinary *roundish sac*, or even a mere *appendage* to the heart, which communicates with the cavity, by means of an opening, corresponding in size to the cavity itself, or in some cases by a narrow aperture, or even by a short canal. This appendage either rises above the cavity of the heart from a broad basis, or rests upon it by means of a neck-like constriction. The *size* of the aneurism varies from that of a pea, a bean, or a nut, to that of a hen's-egg, or of the fist, or may be even larger. The form and size of these structures no doubt mainly depend on the extent and depth to which the metamorphosis of the muscular substance affects the walls of the heart, on their duration and locality, on the patency of the openings in the cavity of the heart, and on the original degree of tenseness and capacity for resistance in the walls of the aneurism. It is probable that the size of the aneurism will be the greater, and that it will the more nearly approximate to the form of a true sac or appendage, in proportion to the extent and penetration of the inflammation of the muscular substance, to its duration, its exposure to the action of the blood flowing into the heart and entering its own cavity through the action of the still uninjured muscular substance, to the degree of contraction affecting the openings in the respective cavities of the heart, and to the yielding of the adventitious tissue constituting the *walls* of the aneurism. Instead of coalescing with the pericardium, as is usually the case, large aneurisms have occasionally met with, adhering directly to the thoracic wall and the lung.

The walls of this form of aneurism of the heart consist, as has been already remarked, of a fibroid tissue, which having taken the place of the muscular substance, coalesces internally with the thickened endocardium. The walls of this form of aneurism never present the slightest trace of a separation of continuity, either in the endocardium alone, or simultaneously in it and in one of the contiguous layers of the muscular substance; for the thickened investment of the aneurism occupying the place of the endocardium always extends beyond the boundaries of the aneurism to the normal wall of the heart, where it is gradually lost in the normal endocardium. The assumption that there is a *lesio continui* in the endocardium may have originated in the circumstance that the endocardium of the cavity in question is frequently found to be hypertrophied, that is to say, it presents several newly deposited layers, the most recent of which extend to the limits of the aneurism, which is filled with fibrinous coagula. The occurrence of an excessive morbid formation of the endocardium on the lining membrane of the vessels, from the blood, as shown in diseases of the arteries, must necessarily, from its great extent and importance, lead to future investigations. This form of aneurism of the heart corresponds to *true aneurism* of the arteries.

The walls of these aneurisms vary in thickness, although they are always thinner than the neighbouring uninjured wall of the heart. They probably become so attenuated, in proportion to the increasing size of the aneurism, as to appear as if they were merely formed by the contact of the endocardium and pericardium, or of a doubled endocardium, in consequence of the aneurism having been developed towards another cavity of the heart. Osseous concretions, especially in the form of laminae, are frequently developed in the tissue constituting the walls of the sac, whence the aneurism acquires a partially osseous character. (See p. 194.)

The cavity in this species of aneurism is very frequently filled with tough stratified fibrinous coagula, as in aneurism of the arteries. This, however, is usually the case only in larger aneurisms, and in fact the mass of fibrinous layers will, in general, be proportional to the size of the aneurism and to the extent to which the muscular fibres are destroyed.

The inner surface of these aneurisms of the heart occasion-

ally exhibit the ordinary villous, shaggy, and warty, or even the so-called globular vegetations.

It is, moreover, worthy of remark, that new layers of endocardium are frequently found to be deposited in great numbers, and to a considerable thickness, upon the inner surface of the aneurism. It is only on a close inspection that they can be detected lying upon the subjacent fibroid tissue. The atheromatous disintegration they occasionally exhibit, imparts a certain degree of importance to their presence, since this atheromatous process is, in some instances, the cause of the origin of aneurism of the heart.

These two species, comprising an acute and chronic form, both of which depend on inflammation, embrace the numerous observations we ourselves have made, and will very probably, on an unbiassed inquiry, be found to include all cases on record.

That inflammation is the original controlling process in this affection is proved not only by the history of inflammation of the muscular tissue generally, and by that of the muscular substance of the heart in particular, but receives additional confirmation from the concurrence of the aneurism with endocarditis and its products, both as to their position and situation, even beyond the limits of the aneurism, generally even as far as the valves,—from the almost universal and simultaneous occurrence of the products of pericarditis,—from the nearly exclusive occurrence of aneurisms in those portions of the heart, which are in like manner the exclusive seat of carditis and endocarditis, viz., the left ventricle,—and from the residua of the secondary metastatic processes in the capillary system, which are frequently of simultaneous date with the endocarditis.

We see no grounds for concurring in the opinion of many observers who regard the alteration of texture of the endocardium and the substance of the heart on which the second form of aneurism depends as a peculiar or unintelligible alteration.

The question here arises, whether an abscess of the heart, (*cardite ulcérate* of Bouillaud,) after opening into one of the cavities, can give rise to the formation of an aneurism. We are of opinion that aneurism of the heart does undoubtedly supervene, but the question is whether such an aneurism is of a persistent character. We have ourselves observed no case corroborative of such a view, and we doubt whether the

pyæmia induced by the opening of the abscess could lead to the consolidation of its walls; that is to say, could heal the abscess by converting it into an aneurism. (Compare pp. 194, 195, on abscess of the heart, and the observations referring to the first form of aneurism of the heart.)

Moreover, we cannot suppose the so-called *atheromatous process*—as it occurs in the arteries—to be the primary condition giving rise to aneurism of the heart. We have already remarked, under the head of hypertrophy of the endocardium, that the newly deposited layers of endocardium upon the wall of the heart have never, in any case that we have observed, been the seat of any but the earliest stages of that metamorphosis which terminates in atheromatous disintegration, while it is only in some instances that it is observed in a more advanced form in the valves. It is, however, worthy of remark, that we have certainly seen this atheromatous disintegration affect some portions of the depositions lining the inner wall of the aneurism, although in a form which proved that it could not, as a primary disease, have given rise to the formation of the aneurism, but must apparently have been subsequently developed in the already existing aneurism.

Aneurism of the heart *occurs almost exclusively* in the left side, and is incomparably more frequent in the left ventricle. There is only one undoubted case on record of this form of aneurism of the left auricle,—namely, that of Chassaignac, to which, however, we must add a preparation in our pathological museum of an aneurism of the acute form in the auricular septum. Hence these formations chiefly occur in the arterial half of the heart, which is known to be the almost exclusive seat of endocarditis and carditis. Like the above named processes, aneurism rarely affects the right side of the heart; and the few cases on record of aneurism of the right ventricle, and those described as situated in the right auricle, prove (like those affecting the left auricle) not to be, strictly speaking, true aneurisms of the heart. These cases usually consist in a general dilatation of the auricle, whose walls have been transformed by inflammation into a fibroid, callous, and even ossifying tissue of considerable thickness, and which itself adheres to the pericardium, having its cavity more or less completely filled up with fibrinous coagula.

In the left ventricle the apex is the ordinary seat of aneu-

rism, and here it also attains its greatest size. It is less frequently observed towards the base of the ventricle, and is of very rare occurrence at the septum, which, however, is commonly more or less implicated in those cases where the apex is the main seat of the disease.

It is only in rare instances that we meet with more than *one* aneurism, and where two or even three exist simultaneously, they are generally in close proximity to one another, and not unfrequently present the appearance of one single aneurism, which has been more or less perfectly separated into two cavities by the marginal elevation of its walls.

That portion of the heart which is affected by aneurism is found, in almost all cases, to be also the seat of an active dilatation, occasioned by the cardo-endocarditic process and its sequelæ, by the aneurism itself, and simultaneous valvular affections.

The spontaneous rupture of an aneurism of the heart may be mentioned as an extremely rare *termination* of the disease. It may open into the cavity of the pericardium, into the pleura, or into the arterial trunk of the opposite cavity of the heart. Such an opening may occur in a chronic form of the disease as the final result of the increasing attenuation of the walls of the aneurismal sac in consequence of its own enlargement, as in a case we have observed where an aneurism in the ventricular septum, and near the apex of the heart, opened into the cavity of the right ventricle, (the *varicose aneurism* of Thurnam.) Acute aneurism of the heart more frequently terminates in laceration, which, in most cases, is very probably induced by the same cause which gave rise to the aneurism itself, namely, inflammation and inflammatory loosening of the tissue, and suppuration in the muscular substance of the heart.—In the case of a boy aged nine years, who presented extensive dilatation and hypertrophy of the left ventricle, and thickening of the endocardium at the septum, we observed immediately below the aortic valves, at the uppermost part of the septum, a laceration about the size of a pea, which led to a sac as large as a nut in the auricular septum, that had been formed by the upheaval of the muscular substance, and after penetrating into the right auricle, opened into its posterior side through an aperture about as large as a hemp-seed.

Most of the cases of aneurism of the heart have been observed in persons of mature age and of more advanced life. When, however, we bear in mind that by far the greater number of cases on record belong to the chronic form of aneurism, we are led to conclude that the disease of the tissue, which is the precursor of the aneurism, must have originated many years before the fatal termination of the disease, and therefore in an earlier period of life, the more so from the circumstance that an appreciable number of cases occur before the age of thirty, while we have observed the acute form in early childhood.

c. Metastasis in the Muscular Substance of the Heart.—Metastatic, purulent, and ichorous abscesses in the muscular substance of the heart may be reckoned among the ordinary conditions giving rise to metastasis, more especially when occurring in consequence of pus or ichor being taken up into the mass of the blood. There are usually several of these abscesses present, and they may result in laceration or ulcerous perforation of the heart. Metastatic processes are always simultaneously present, to a considerable extent, in other organs.

d. Gangrene of the Heart.—There is nothing, *à priori*, at variance with the possibility of the occurrence of gangrene in the muscular substance of the heart. Ulcerations accompanied with malignant products are not of rare occurrence, but the correctness of the observations purporting to refer to gangrene of the heart, have nevertheless been called in question by several writers, and we must remark that no case of the kind has fallen under our notice.

e. Adventitious Products.—Although adventitious products, with the exception of the adventitious tissue developed from inflammation, are generally of rare occurrence in the heart, yet some forms are not unfrequently met with; as, for instance, the varieties of adiposity of the heart, which we now proceed to notice.

1. *Adiposity of the Heart.*¹—The occurrence of fat in the heart presents various anomalies, and exhibits different degrees and forms. (Compare the observations made on fatty degeneration of the Muscles, in Vol. III, pp. 312—15.)

a. The first form consists in the accumulation of an unusual quantity of fat on the surface of the heart. Fat is generally

¹ Oesterr. Med. Jahrbücher, B. xxiv, St. 1.

first abnormally deposited in those parts which, in their normal state, are covered by a certain quantity of fat, even in general emaciation; as the base of the heart, the sulcus transversalis, around the point of origin of both the arterial trunks, the sulcus longitudinalis and the course of the coronary vessels, the margin and anterior surface of the right ventricle, and the apex of the heart. The right side of the heart is always covered with large quantities of fat whenever there is any considerable tendency to the production of this tissue. In some cases this formation of fat is so excessive as to enclose the whole heart in a thick irregularly lobed mass of adipose matter, giving it the appearance of being enlarged.

This accumulation of fat in the heart is usually associated with a similar accumulation in the pericardium, in the mediastina, and in the abdomen, that is to say, in the omentum and mesentery, and on the gall bladder, with fatty liver and with general corpulence. The muscular wall of the heart, in young men of great muscular strength, is found to be in a normal condition, but in persons of advanced age, and in females, in conformity with the general character of the muscular substance, it is in general relatively thinner, more flaccid, discoloured, and paler.

The latter condition, which constitutes the transition to a second form of adiposity, and in like manner varies in degree, represents—

b. Actual Fatty Degeneration of the Heart.—Fatty Metamorphosis of the Muscular Substance.—The fat surrounding the heart penetrates inwards, and by gradually insinuating itself between the muscular fibres, tends in this way to displace the muscular substance. The apex of the heart and the right ventricle are especially subject to this form of degeneration, which, according to Laennec's observations, originates at the first of these points. When the left ventricle is implicated, the disease is usually limited to the apex, from whence it advances towards the right ventricle. It is only in its more intense stages that it affects the main part of the left ventricle. The muscular substance at the apex of each side of the heart, and consequently in the right ventricle, is frequently observed to be reduced to a layer, which, from its extreme thinness, scarcely admits of being measured, and appears like

a mere muscular investment covering the fat. In cases of intense degeneration, the muscular wall of the left ventricle has even been found only from 2—1½ lines in thickness. The muscular substance is flabby and much relaxed, of a faded colour, capable of being easily torn, and infiltrated with free fat. This displacement and disappearance of the muscular fibres is similar to the alterations observed in the muscular coat of the intestine when the mesentery is intensely fatty, and in the corresponding coat of the gall bladder. The valves of the heart are at the same time thin and transparent, while the papillary tendons are softened.

This fatty metamorphosis does not only occur in the form of the above-mentioned transition stage, and in consequence of the excessive production of fat, and simultaneously with other accumulations of fat, but likewise independently of any such connection, and accompanied with general emaciation, as the result of tuberculosis and tuberculous phthisis, and lastly under circumstances that have not yet been explained. It is of frequent occurrence in conjunction with fatty liver. It is rarely met with before the age of 30—35 years, and is incomparably more frequent afterwards; it is also much more common in women than in men.

Considerable interest attaches itself to a not unfrequent combination of these two forms of adiposity of the heart, in which there is atheromatous disintegration and ossification of the morbidly deposited layers of the inner coat of the arteries, and especially of the trunk of the aorta, associated with aneurismal formation in the trunk of the aorta. These fatty degenerations are, however, very frequently associated with ossification of the coronary arteries,—a circumstance which will be further considered when we treat of those fatty accumulations on the trunk and extremities, attended with atrophy of the muscular substance, and with ossification of the arteries, which remind us of other analogous combinations of fatty accumulation with formation of bone, as in lipoma, fatty cysts in the ovaries, accumulation of cholesterin in ossifying cysts, &c.

However we might be disposed to imagine that fatty metamorphosis would frequently terminate in spontaneous laceration of the heart, such is very rarely the case, even where the fatty degeneration extends to the left ventricle, which, as

the ordinary seat of spontaneous lacerations, would seem predisposed to this lesion.

c. There is a third and very important, although hitherto unnoticed form (See vol. iii, p. 315) of this disease peculiar to the muscular substance of the heart, and differing entirely from the two previous forms of adiposity.

This form occurs more especially in hypertrophied and dilated hearts, in combination with the residua of endocarditis and carditis, or independently of these. The extent, seat, and duration of the disease present numerous remarkable diversities. In some cases, we observe scattered and distinct centres of inconsiderable extent, where the muscular substance is pale, flaccid, of a dirty yellow colour, and soft and friable, rather than admitting of being easily torn, as is usual in relaxation. In other cases these centres are very numerous, and are found scattered over the true substance of the heart, in the trabeculae, and in the papillary muscles. They are ill defined, their margins being indistinct or obliterated. The discoloration presents a striped appearance as it follows the course of separate muscular fibres, decreasing in intensity from the centre outwards, and being finally lost in the normal colour of the substance of the heart. This anomalous condition frequently extends over the whole inner layer of the muscular substance, which, when seen through the endocardium, after it has become thin and even transparent, presents the discoloration to which we have already referred, showing, on a closer inspection, that this change of tint depends on the presence of fine yellow granules or globules, which are deposited in great numbers, in close contact, as if strung together on strings, in and upon the muscular substance, and variously entwined among the muscular fibres. The trabeculae and the papillary muscles are usually diseased throughout their whole extent, as is also the muscular wall of the heart through its entire thickness, although not uniformly in all parts. This granular formation on and between the muscular fibres marks the intensity of the disease, which further corresponds with the degree of discoloration and softening of the muscular substance. A microscopic examination shows an accumulation of black and dark-outlined globules, which prove to be fat, while the muscular fibres are found to have lost their striated appearance, and

the fibrilli are soft, and readily break down into delicate molecules.

This form of adiposity most commonly occurs in the muscular substance of the left ventricle, and, in cases of hypertrophy, also in the right ventricle.

This affection is, according to our observations, the most frequent cause of the *spontaneous laceration* of the hypertrophied left ventricle.

It may, moreover, probably be regarded as a consecutive disease of hypertrophy of the heart, since it is developed in consequence of the state of paralysis or inertness of certain portions of the muscular substance, induced by the disproportion between the mass of the tissues and the powers of innervation. The conditions of this disease are therefore similar to those experienced by the voluntary muscles in fatty metamorphosis (the second form of adiposity). We have occasionally observed this form in hypertrophied muscular membranes, when the paralytic habitus is established, as, for instance, in the hypertrophied muscular coats of the intestine and the bladder.

We have, however, occasionally met with this form of disease in non-hypertrophied hearts in young persons. Dilatation had probably been induced here by the adiposity, and the muscular substance relaxed in consequence. It is highly worthy of notice, that the papillary muscles are sometimes especially, and very extensively, diseased, as they may give rise to endocarditic murmurs and insufficiency of the valves, in consequence of inefficient action and tension.

2. *Cysts*.—These formations are very uncommon in the muscular substance of the heart, especially if we refer to cysts containing entozoa. In treating of them, we will limit ourselves to acephalocysts, deferring all notice of the cysticercus till we consider entozoa.

A sac containing acephalocysts is very rarely met with in the muscular substance of the heart, there being only a few cases on record, to which, however, we must subjoin two derived from our own observation. The parent sac contains either one or many *acephalocysts*. In one of the cases, we observed only one acephalocyst, which almost entirely filled up the cavity of the parent sac; while in the other, the parent

sac, as far as the injured condition of its contents enabled us to judge, was filled with many of these cysts. We are induced, from the rarity of their occurrence, to give a short report of these cases, the former of which was rendered peculiarly interesting from the circumstance, that the presence of these acephalocysts occasioned sudden death. A short notice of the second case, in one of the medical journals, is the only account that has yet been published of either of these two cases.¹

1st CASE.—This case refers to a young woman, aged 23 years, whose sudden death led to a judicial inquiry. The heart was somewhat enlarged and hypertrophied. The uppermost part of the ventricular septum presented a fibro-serous cyst with delicate walls, and larger than a hen's egg, which protruded into both ventricles, but more especially into the right and the conus arteriosus, and had so thoroughly displaced the muscular substance, as to be almost exposed. It had burst over an extent of $1\frac{1}{4}$ " towards the right ventricle. From this opening an acephalocyst, nearly equal in volume to the parent sac, had been thrown with the blood into the conus arteriosus and the pulmonary artery, where it was found tightly wedged, and so far within the trunk of the artery as nearly to reach to its left branch.

The liver in this case was very large, and the right lobe contained *one* acephalocyst of the size of a child's head, and *two* of about the size of a hen's egg.

2d CASE.—The heart of a soldier, aged 35 years, was examined; his sudden death being made the subject of judicial inquiry, as in the former case. The posterior and uppermost part of the ventricular septum, and the contiguous portion of the posterior wall of the left ventricle, were occupied by a round sac of the size of a duck's egg, having callous walls of a line in thickness, which projected into the cavities of the right ventricle and auricle. Towards the back of the sac the muscular substance of the heart had disappeared, while the heart itself was attached at that point to the pericardium by a dense cellular tissue. On making a section through the wall of the sac, a rust-coloured stripe was observed between an outer and inner layer of white fibroid tissue, this stripe being the remains of the peripheral

¹ Oesterr. med. Jahrbücher. Jahrg., 1841. Juni.

coagulum of a hæmorrhagic exudation. The sac contained a pulpy brown fluid intermixed with crumbling and shaggy fibrinous coagula and the soft gelatinous remains of acephalocysts.

3. *Fibroid Tissue*.—Fibroid tissue very frequently occurs in the form of a fibroid thickening of the endocardium on the inner surface of the heart, as a fibroid thickening of the valves and their tendons, in the heart's walls, and in the tissue of the papillary muscles and the trabeculæ, where it is accumulated in different quantities and forms. We have always found that this product was based on some inflammatory process—endocarditis, carditis, or their combination.

It appears in the muscular substance of the heart, either in the form of white stripes, of diffused strata of various thickness, or finally of large, roundish nodules, or irregularly branching masses. This substance, moreover, constitutes the greatest portion or the whole of the walls of aneurism of the heart, when of a chronic form.

We have never observed fibroid tissue in the heart under the form of an independent fibrous tumour.

4. *Anomalous Osseous Substance*.—Osseous structures are frequently found within and upon the heart in the form of bony concretions. They invariably originate in the fibroid tissue which is produced, as we have already mentioned, by inflammation, and in the deposition of new layers of endocardium. In this manner bony concretions are occasionally developed in the fleshy wall of the heart, and in the tissue of the papillary muscles and of the trabeculæ, in the form of nodular uneven plates, of nodular bands, or of irregular ramifying osseous masses. In the valves where these formations are of frequent occurrence, they are often in the form of nodular, ramifying bands and rings of different thickness. The whole fibroid mass sometimes ossifies, and may then be seen lying free and uncovered in the cavity of the heart, both in the valves and on the wall of the heart, when the muscular fibres are completely destroyed. Bony concretions in the valves are not unfrequently connected with others in the tissue of the heart, and (as we sometimes remark in diseases of the pericardium,) with osseous formations in pericarditic exudations of fibroid texture; or lastly, they form a bony mass, branching out in various directions. Smaller cylindrically-shaped concretions are often met with in

the thickened tendons of the papillary muscles. In ossification of the valves these cylinders are larger and more connected.

The valves of the left side of the heart, especially the aortic valves, also exhibit osseous formations, which are developed in the morbidly deposited hypertrophied endocardium. In the auriculo-ventricular valves they form plates of inconsiderable size, and in the aortic valves string-like or nodularly rounded concretions. They are distinguished from the bony formations produced from the fibroid inflammatory callus by their yellow colour and their similarity to ossifications of the arteries.

We have never been able fully to satisfy ourselves in reference to this last-named osseous formation upon the endocardium of the wall of the heart.

Fibroid tissue and the bony concretions into which it is developed, when they are the remote consequence of inflammation of the endocardium or the tissue of the heart, are limited, like this process itself, almost exclusively to the left side of the heart. The few cases in which they are observed on the right side of the heart are probably those in which there is ossification of the valves. The occurrence of this osseous formation in the left side is moreover limited to the valves and the ventricle, and never extends, according to our observations, to the left auricle. When bony formations occur in an endocardium which presents a morbid deposit, they are almost exclusively limited to the left side of the heart.

5. *Tubercles*.—If we except those cases of tuberculosis which have originated in the neighbouring tissues and have extended to the organic muscular coats of other structures, as the intestine, &c., tubercles occur in the substance of the heart with the same rarity as in muscle generally. It is only in extreme degrees of tuberculosis that we have discovered one or more tuberculous masses in the muscular substance of the heart in addition to a tuberculous exudation on the pericardium. We must, however, here except those cases in which large tuberculous masses, exuded on the external investment of the heart, have gradually imbedded themselves in the outer layer of the muscular substance.

It is remarkable, considering the similarity of the process of exudation on the endocardium and on the inner coat of the

vessels with that on serous membranes, that tuberculosis should not occur on the two first-named structures.

6. *Cancer*.—Cancer of the heart is an extremely rare disease, and its occurrence is, probably, invariably owing to a highly developed cancerous dyscrasia, or to the proximity of a cancerous formation, as for instance in the mediastinum. The form of cancer affecting the heart appears, as far as we know, to be limited to medullary cancer in its genuine type, or in the form of melanosis. It is developed under and in the external investment of the heart, in any portion of the fleshy walls, or immediately below the endocardium, protruding, according to its size, more or less extensively either inwards or outwards, or even in both directions, in the form of nodules and clumps.

We have observed a case of acute medullary cancer of the heart accompanied by very general acute cancerous formation, in the form of numerous, small, roundish nodules, seated in the innermost layers of the substance of the heart beneath the endocardium, and even upon it, somewhat in the manner of globular vegetations. The above general observations will show that this mode of formation of cancer must be regarded as a highly interesting form of disease of the fibrin.

Cruveilhier (Livr. 29) has described the case of a ragged cancerous tumour seated on the inner surface of the right auricle, and projecting into the cava descendens and the right ventricle. This cancer probably resembled primary cancer of the veins.

7. *Entozoa*.—In addition to acephalocysts, to which we have already referred, the *Cysticercus* is by no means of rare occurrence in the heart, being then also simultaneously present in some of the voluntary muscles. There are seldom more than one or a very few of these worms to be found together in the substance of the heart; and in these cases they are also commonly present in the brain.

The *Trichina*, unlike the *cysticercus*, does not occur in the heart, although it exists in the voluntary muscles.

In addition to the above named secondary formations we will here notice certain morbid structures which appear in the cavities of the heart, either free or adhering. These are not products of the endocardium, but essentially fibrinous concretions from the blood, and differing, therefore, from the above

secondary formations, both in this and other respects. They constitute a series of formations which we will consider under the following title :

8. *Coagula, Polypi, Vegetations in the Cavities of the Heart.*¹—The above terms have, at different times, been applied to this class of concretions. We refer our readers to the general remarks on the diseases of the blood and its fibrin for all that relates to the pathology, nature, and metamorphosis of these structures. We purpose here treating specially of their form, and shall only touch upon their other relations as far as is necessary towards the right comprehension of a subject which has been much beset with errors in the present day.

The structures now under consideration occur in many forms. The question here arises as to these structures generally, and each form specially, whether they are produced after death or before it, and how long they had subsisted during life. It has long been customary to distinguish certain fibrinous coagula from others by the designation of *death-polypi*. The fact that the blood, in consequence of the arrest of the heart's action, coagulates more or less perfectly in the cavities, into a loose soft clot, or a more compact mass, from which the fibrin is more or less thoroughly separated into a concretion, which, in its turn, exhibits the most various degrees of consistence or plasticity, has long been regarded as entirely in unison with the phenomena observed in drawn blood that has been left to stand and cool. The symptoms presented in the course of disease, the peculiar character of the phenomena exhibited in the death-struggle, the form of certain fibrinous structures in the heart, their relation to its inner surface, and their adhesion to the endocardium, have long since been advanced in support of the view that there may exist, during life, an independent self-persistent polypus of the heart. This view has continued, to our own day, to be so entirely misunderstood and misapplied that even ordinary death-polypi have very commonly been mistaken for true polypi of the heart.

No doubt can be entertained in the present day that fibrinous concretions are formed in the heart from the blood during life. It would appear certain that they form an

¹ Oesterr. med. Jahrbücher, B. xxiv, St. 1.

organic (textural) connection with the inner wall of the heart; and further, that they experience various metamorphoses in their elementary composition. We purpose considering these in the sequel; but we would, in the first place, make a few general remarks on the conditions under which fibrin is separated during life from the blood, and coagulates into different forms of concretions. We will also consider, under the head of these respective forms, all that relates to their formation after death or during life.

These conditions exist partly in the heart and partly in the blood, and both are not unfrequently co-existent: the latter, however, are the more important, while the former are to be regarded as merely affording favorable momenta.

1. The first condition involves an abnormally prolonged continuance of the blood in the cavities of the heart in consequence of a decrease in the activity of the heart's action, as in hypertrophies of considerable intensity, passive dilatations, aneurisms of the heart, and in every death-struggle depending on general paralysis, or in consequence of pre-existing contractions (stenoses) of the ostia; or, lastly, there may exist various mechanical conditions in the form of inequalities and roughnesses on the inner surface of the wall of the heart and on the valves, in its passage over which the blood deposits its fibrin in a corresponding form.

2. The other and most essential condition consists in the tendency of the blood to coagulate or to part with its fibrin in various forms of coagulation, either in consequence of spontaneous disease, or of the absorption, in various ways, of some heterogeneous matter. Under this head we must especially class the so-called inflammatory (croupous) crasis, as it occurs in a primary or secondary form, associated with inflammations, pneumonia, rheumatism, &c.; the poisoning of the blood by the absorption of the multifarious products of the inflammation of normal or abnormal tissue, which have been produced within the vascular system, on the endocardium, on the lining coat of the vessels, or externally to the vascular system, having in the latter case reached the blood by the most various channels.

The coagula in the heart may be classed in the following order in reference to their form. Many have only recently

been recognised and duly characterised as fibrinous concretions, and these have received designations corresponding to their forms.

a. *Clotty, roundish, membranous, ramifying coagula*, when occurring in the cavities of the heart, commonly receive the designation of *polypi* or *polypous coagula*. These are variously sized clots, presenting differences in the number and length of the ramifying appendices by which their rounded forms are modified. They consist of a dark or blackish red clot, from which fibrin is separated at some portions of the periphery, but seldom from the interior; or they consist, for the most part, of fibrin which has absorbed a certain quantity of cruor and serum, and appears coloured with various tinges of red, or, when free from these, it exhibits a pale and somewhat dense coagulation. These concretions are especially common in the right side of the heart, where they are found in large quantities, generally associated with a loose coagulum and fluid blood, and exhibiting coagula which have been formed during the last moments of life and after death. This form of coagulum does, however, undoubtedly occur at various periods before death. Without entering into a description of these coagula, the limits of which it is difficult to separate from those of the first-named variety we would merely remark, that the following conditions favour their development during life:

1. When they are situated in the left half of the heart, especially when extending into the aorta and its branches.
2. When their ramifications extend into the ventricles, and their branches are entwined among the trabecule and the tendons of the papillary muscles.
3. When they exhibit the impression of the contiguous surface of the heart, as is especially manifested in the auricular appendage (the auricle proper).
4. When they adhere or coalesce with the inner surface of the heart—the endocardium.
5. When they consist of pure fibrin, and are at the same time tough and tenacious.
6. When they exhibit a dirty-yellowish and greenish colour, and are, moreover, opaque.
7. When they present small purulent foci or tuberculous concretions.

8. When any one of the associated diseases of the blood is developed.

These coagula cannot in themselves be regarded as symptoms of endocarditis where other essential evidences of the presence of this disease are wanting; and even where the latter are present, they cannot be considered as affording any direct proof of the existence of the endocarditic process, but simply of a pre-existing and spontaneous disease of the blood, depending, probably, on the absorption of endocarditic products.

It is incontestible, that these coagula not only adhere to the endocardium, as has been observed, but that they are also capable of entering into an organic or textural connection with the lining membrane of the heart, and thus vegetate independently and without the aid of a vascular system, as they are directly surrounded by the liquor sanguinis. On submitting the observations hitherto made on this subject to the severest criticism, we meet, amid a mass of erroneous and hasty conclusions, with some few cases which unquestionably belong to this class of coagula. These tumours are of various size; of a roundish, oval, cylindrical form, which appear attached to a broad or narrow base, as by a pedicle; they are of a sponge-like, or elastic and tough, consistence; they generally consist of a fibroid structure, but in some cases exhibit a soft texture, composed of membranes, covered with elementary granules and cellular nuclei, of delicate fibrilli, and of thick and even tubular fibres and amorphous coagula. Their colour is red, yellowish red, or white. When they are gradually receiving one or more coverings of newly formed endocardium, which extends from them to the inner surface of the heart, they present the appearance of having been developed below the original endocardium, or, at all events, in the innermost layers of the muscular substance of the heart. Osseous and cretaceous concretions may be successively developed in them; and the free stony concretions, which former observers have recorded as occurring in the cavities of the heart, were, doubtless, nothing more than loosened, liberated, fibrinous coagula, which have become ossified or cretified. We have never hitherto been able to detect vessels in them. (See Faber, Thomson, Vernois.)

It is probable, that these coagula are somewhat diminished

by a process of solution upon their surface, before they acquire any decided texture and are covered by layers of endocardium, and that they thus lose their original form, which is probably an irregular one, and become round. Such a supposition seems to derive support from analogy with the disappearance of vegetations on the valves of the heart, the gradual rounding of the globular vegetations, and the diminution and disappearance of the plug in an artery after the application of a ligature.

b. *Globular vegetations* (*végétations globuleuses* of Laennec) in the cavities of the heart constitute a second form. The formations distinguished by this designation are generally round concretions, varying from the size of a pin's head to that of a nut, attached by means of ramifying, cylindrical, or flat appendages or bands which entwine themselves among the trabeculæ of the heart; and are of a more or less uniformly dirty, greyish-red, or white colour. They are hollow in the interior, but contain, within a wall of irregular thickness, a dirty greyish red, or even chocolate coloured thickish fluid, resembling cream or pus, and which is occasionally of a dirty whitish or yellow colour. One or more of these concretions very frequently burst, when the fluid may be seen effused into the cavity of the heart, and distributed over the recent coagula which have been formed either in the death-struggle or shortly after death; or it is found mixed with the fluid blood contained in the cavity. The band-like appendages which they throw out, are either solid, or softened and liquefied in their interior.

Besides the structures of this form, there are others belonging to the same class which exhibit different relations, being of an oval shape, somewhat like a wedge, and presenting a shaggy or villous appearance. They differ from those already named, by adhering directly and firmly to the endocardium.

Although we very commonly meet with these structures in the condition above described, this state is not the primary one in which they occur, but merely the result of a metamorphosis to which the fibrinous coagulum has been subjected, not only in its elementary character, but also in its external form. Cases may be occasionally met with in the course of a long-protracted series of observations, in which this metamorphosis may be followed through all its gradations.

The globular vegetation is originally a solid fibrinous coagulum of irregular form, which varies in colour according to the number of blood-corpuscles it contains, from different shades of red to a reddish white colour. This coagulum gradually assumes a roundish form, probably in consequence of the outer portion being taken up in the blood in a finely comminuted state. The metamorphosis which it undergoes is very important, and begins as a softening disintegration or solution in the interior of the nucleus, from whence it extends towards the surface. This process is so far developed in the globular vegetations above described, that there only remains a peripheral layer, which encloses the dissolved part as in a capsule. The soft and diffuent mass consists, as has been already remarked, of a pulpy, cream-like fluid, very often resembling pus, and of a chocolate, or dirty brownish red, reddish grey, pale yellow, or whitish colour. A similar metamorphosis affects the ramifying band-like coagula, proceeding from the vegetations when they become hollow. The same process is occasionally discernible in the central layers of those coagula of the first form which have arisen during life; we sometimes observe in these coagula a tendency to decomposition, both by their turbidity and opacity, their dirty-yellow colour, their extreme lacerability, and by the appearance of a turbid cream-like moisture when they are compressed and torn.

This metamorphosis of the fibrinous coagulum is, moreover, highly interesting, from the numerous and important analogies it presents. It is here undoubtedly dependent on disease of the fibrin, from which the coagulum itself is formed, as we have invariably observed these globular vegetations only in cases in which the blood is in a state of dyscrasia, as in *croupous* processes, after typhus, in the pyæmia of phlebitis, in a similar condition of the blood in the course of tuberculous or cancerous disorganisation, &c.

It is a remarkable circumstance that globular vegetations are almost always limited to the left ventricle, where they are attached in the manner already described to the apex and the contiguous parts. We have, however, observed a few exceptional cases in which globular vegetations were situated in the left auricle as well as in the right ventricle and auricle.

A proof of the part contributed to their formation and

attachment by mechanical conditions is afforded by the fact of their being deposited in the apex of the left ventricle, and in the appendages of the auricles—in short in those parts of the cavities of the heart which are most favorable to stagnation of the blood. We have seen these formations in the cavity of an aneurism seated at the apex of the left ventricle, and also, together with the ordinary valvular vegetations, on the mitral and the aortic valves in endocarditis.

While, on the one hand, every fibrinous coagulum, when considered in reference to its most essential feature—its metamorphosis—may be transformed into a globular vegetation, and coagula of the first form may thus be converted into these globular structures, there can, on the other hand, scarcely be said to be any true limits between the globular vegetations and those on the valves of the heart. The latter not only very frequently assume the globular form, as will be seen from the following remarks, but valvular excrescences pass through the metamorphosis of globular vegetations without assuming this form.

c. The third form comprises all those coagula that have in recent times been distinguished under the collective designation of *vegetations of the valves of the heart*. These were formerly known as *sarcomatous, fungous, condylomatous excrescences of the valves of the heart*, and have derived especial importance in our own day in consequence of being commonly regarded as an infallible criterion of endocarditis. This form, which is more frequent than either of the others, also presents the greatest variety in reference to number, bulk, shape, mode of attachment, colour, consistence, and internal composition.

The *form* of these vegetations is partly influenced by their mass or size.

Smaller vegetations occasionally exhibit a superficial *roughness*, only appreciable to the sight and touch on a close investigation, and which is produced by the presence of *fine granular or extremely delicate villous* structures on the endocardium of the valves.

When these structures are deposited upon one another in a finely granular form, they are more prominently visible on the surface of the valves.

They commonly present a *coarsely granular or villous* and

finally a *shaggy* appearance, measure several lines in length, and are arranged either in rows of *rigid*, pointed, unyielding *excrecences*, or soft, relaxed, and *pendent villi*.

They form *shaggy appendages*, having a thick, club-like, free extremity; or, when of a more considerable size, they form round, oval, or pyriform *pedicled excrecences*.

Lastly, when of considerable dimensions, they somewhat resemble condylomata, having a *cock's comb* or *mulberry-like appearance*, or they are irregularly *nodular*, and either broad or pedicled.

Partial reference has already been made to the *dimensions* of these vegetations, which vary from the size of a hemp-seed to that of a hazle-nut.

As we have already remarked, these structures may occur in very *small* or in very *large numbers*. In the latter case different forms and sizes are usually found associated together; at the same time, they are commonly spread over a considerable extent of surface.

Their *colour*, consistence, and composition vary according to their age, and the quality of the fibrin from which they are formed. We shall, however, revert to this subject in the proper place.

Their principal *seat* is in the valvular apparatus; they attack the mitral as well as the aortic valves of the left side of the heart, and are generally remarkable for the number and size in which they are exhibited in all the different forms of this affection, to which we have already referred. They are, moreover, observed on the tendons of the papillary muscles—in any part of the inner surface of the heart, (the endocardium of which is, in consequence, thickened and rendered opaque, while its surface presents an absence of smoothness,)—in and upon the margins of any fissure of the endocardium or of the subjacent tissue—on the margin of a fissure in the valve—on the edge of acute aneurism of the heart—on the torn extremities of a papillary tendon—on the inner wall of chronic aneurism of the heart—and, lastly, even without the heart, on rough, ragged, and uneven spots on the inner surface of the arterial trunks.

They occur especially on the valves in small numbers, in the form of minute granular or villous depositions at the

separate segments of the auriculo-ventricular valves, or on the nodules of the semilunar valves, and in their vicinity. They, moreover, in some cases form a granular, villous, or shaggy margin of varying breadth, near the free edge of the valve, which, inclining in a crescent-like form along the semilunar valves, follows the fibrous coat in the parenchyma of the valves. When occurring in great numbers, they occupy a considerable portion of the free margin of the valve, and, assuming every possible form, extend upwards over the whole valve to the endocardium of the auricle, and downwards to the tendons of the papillary muscles.

At other portions of the endocardium they commonly form granular or delicate villous deposits at the margin and in the vicinity of fissures, and most frequently near some exuberant quantity of large villous masses.

It is worthy of remark, that all these forms of vegetations follow the course of the blood-current in every direction. Where they exhibit a broader margin on the auriculo-ventricular valve, this margin forms a projecting angle, from whence it is rapidly deflected. When they form villous or larger masses, they incline at the auriculo-ventricular valve towards the ventricle, and at the semilunar valves towards the direction of the vessel. We must also observe, that they are always situated on the side of the valve which is turned towards the calibre of the implicated opening.

In reference to their *colour*, they are, when newly formed, and at the commencement of their existence, usually of a pale blue or yellowish-red colour, less frequently dark red, and are either uniformly coloured, or speckled and seamed. They gradually become pale, resembling faint yellow, faded, and thoroughly washed fibrin; frequently, however they do not part with their hæmatin, which in its further metamorphoses gradually loses its colour, assuming a brownish red, rusty yeast-like tinge, by which the vegetation is permanently characterised. These structures usually exhibit the consistence of a fibrinous coagulum, varying in their degree of softness or hardness; thus they usually become harder in proportion to the increased paleness of their colour, although in some rarer cases they are soft, dissolving like the globular vegetations.

On lifting or tearing off the vegetation, there immediately

appears, if it be recent, a loosened, excoriated, and rough portion of the endocardium, which, in structures of older formation, is also raised and swelled up. As they become older, they at the same time become more firmly attached to the endocardium.

The following facts may be noticed in reference to their metamorphoses subsequent to the process from which they originate :

a. Vegetations once formed, in most cases, remain stationary for a long time, or even through the whole period of life, more especially when they have acquired any considerable dimensions ; but it is certainly undeniable that they may, in the course of time, shrink and diminish, and exhibit an increase of condensation and consistence (*excroissances cornées, cartilagineuses*, Bouillaud), as we see in other fibrinous coagula.

b. There is no doubt that they diminish in a different manner, and that at times their presence is scarcely perceptible, since they often degenerate into fine, whitish, brush-like fibrinous villi, and in some cases even wholly disappear, without leaving any trace of their existence. The latter is proved by the circumstance (See p. 184) that, while in obsolete cases of endocarditis, the valves exhibit very insignificant or even no traces of vegetations, notwithstanding that they bear the impression of former intense disease, recent endocarditis very commonly presents a large number of these structures, characterised, in many cases, by the size and quantity in which they occur. These vegetations present an analogy with other fibrinous coagula within the vascular system, by being worn out, as it were, superficially, that is to say, they are taken up into the blood in fine particles, and are thus gradually diminished. This remark especially refers to such vegetations or portions of them as are separated from the fibrin of the blood of the heart in the form of coagula, whilst those which have been deposited by exudation remain and shrivel up. (See our subsequent remarks on the origin and nature of these vegetations.)

c. These vegetations on the valves—in perfect analogy with other fibrinous coagula—undergo, although less directly, a bony and chalky metamorphosis, constituting a special form of valvular ossification, to which we will revert in the sequel.

d. These vegetations seldom, and indeed never, unless when of considerable dimensions, experience that metamorphosis of

softening, by which a fibrinous coagulum is converted into a hollow globular vegetation. This metamorphosis, which occurs in the early stages of recent vegetation, is undoubtedly the result of extensive disease of the fibrin.

In conformity with their elementary character, they consist, according to their respective ages, of elementary granules, cell-nuclei, and cells—of a homogeneous base, intersected by nucleated fibres, in the manner of the longitudinal fibrous coat—of fibres and fibrillæ resembling cellular tissue, and of thick tubular fibres.

The corresponding opening is more or less closed, in proportion to their number and volume.

In all considerations that relate to the origin of these vegetations we ought, in the first place, to notice their relation to the endocarditic process.

In the greater number of cases these structures are accompanied with the phenomena of endocarditis—the alterations of texture to which it gives rise; their appearance so far coinciding with these phenomena, that recent vegetations are found simultaneously to occur with recent derangements of texture, and obsolete vegetations with inveterate disturbances of texture—the residua of endocarditis. The question here arises in relation to these cases, *are these vegetations endocarditic exudations? and if not, how can their origin depend upon the process of endocarditis?*

In some rare cases they are observed unaccompanied with any phenomena of endocarditis; and here it may be asked, *how is their origin to be explained, and on what does it mainly depend?*

The results yielded by very numerous and widely differing cases are as follows:

a. That these vegetations, when considered collectively, are, in some cases, *direct products of inflammation*—that is to say, *exudations*.

b. That in the great majority of cases they are *only in part* to be regarded as inflammatory products, since it is only the lowest layer, directly adhering to the excoriated valve, that can be considered in the light of an exudation, whilst *the greater number have been produced in another indirect and secondary manner from the endocarditis*.

c. That they also occasionally occur *without the existence of endocarditis*.

In the two latter cases, the vegetations occur as fibrinous coagula deposited by the blood, and their formation is effected in the following indirect and secondary manner :

Endocarditis induces a diseased condition of the blood, in consequence of the latter taking up its exudations. This morbid state is manifested by the readiness with which its fibrin coagulates and separates. As such coagula occur in different parts of the capillary system (as secondary processes in the spleen and kidneys), so also is the fibrin separated from the blood in the heart with a readiness proportional to the vegetations produced by the endocarditic process in the form of exudations, or the number of loose, rough, felt-like excoriated spots on the endocardium, either of which may exert a mechanical action.

The number and dimensions of these secondary vegetations accord with the intensity of this disease of the blood, and more especially with its character ; and we find that they occur in the most exuberant masses when there is intense endocarditis, manifested by simultaneous disturbances of texture, and still more so where the disease is characterised by suppuration. This correspondence is further manifested in a remarkable manner by the great number of secondary processes in the different parenchymatous structures to which we have referred. The mechanical influence is more developed in proportion to the greater intensity of the endocarditic process. The number of vegetations is, however, most remarkable on the margins of a fissure in the endocardium and in the subjacent tissues, occasioned by inflammatory loosening of the texture, or still more, perhaps, by suppuration. It is obvious that the number of these vegetations increases with the extension of the endocarditis, and of the space over which the mechanical influence has diffused itself.

The metamorphosis of all these vegetations generally, and of those of the second form especially, depends upon an internal cause (namely the blood). Where, as is usually the case, the product deposited is of a benignant nature, the fibrin constituting these vegetations experiences the above named favorable modifications, that is to say, the vegetations become condensed,

gradually diminish, and even wholly disappear, or cretify. This is in accordance with such terminations as shrivelling, obliteration, and atrophy, which usually characterise the secondary process accompanying such an endocarditis. In some less frequent cases the vegetations undergo a softening process, and become diffuent in their interior, yielding a variously coloured purulent fluid. This is observed in intense endocarditis, and when it occurs with purulent exudation, and in this respect it also agrees with those secondary processes of a less benignant character which terminate in purulent fusion. These metamorphoses are more commonly manifested in vegetations consisting of large club-like villi or roundish masses, which, as we have already observed, accompany intense endocarditis, characterised by purulent exudations. The vegetations that begin to dissolve at the centre approach more nearly to the character of the *globular* kind in proportion to the roundness of their form.

The size and the metamorphosis of the vegetations afford evidence of the intensity of the endocarditis, and more especially of the quality of its products, when, besides these, other essential phenomena of endocarditis are present, and when the diseased condition of the blood can alone be referred to endocarditis.

In some rare cases, where these vegetations are unaccompanied by any other important phenomena of endocarditis, they are usually inconsiderable in number and dimensions; and the question might arise, whether they may not even here originate in some very slight degree of endocarditis, which might produce scarcely perceptible disturbances of texture, that had been masked and hidden by the presence of the vegetations. We must, however, bear in mind that the origin of these fibrinous coagula is, in general, mainly dependent on some peculiar character in the blood, that it admits not unfrequently of being referred to some process remote from the heart, and that in some cases even it may be said to be spontaneously developed; that in addition to benignant and inconsiderable vegetations, there are other extensively diffused secondary processes in the different parenchymatous structures, which have a wholly heterogeneous character, and terminate in purulent fusion; that there is no trace of endocarditis to be detected, or, at all events,

no new endocarditis corresponding to the recent condition of the vegetations; that a mechanical influence is especially important in the deposition of these vegetations which are formed not only on every rough part of the endocardium and the valves, but even on the lining membrane of the vascular trunks; and lastly, that the normal valvular apparatus, by means of its tendons, affords a highly favoring requirement for the separation of the fibrin. The above observations leave no doubt that, like other fibrinous coagula, these vegetations may be formed and deposited independently of simultaneous endocarditis, and in consequence of some other disease of the blood, upon any favorable portion of the inner surface of the heart, as, for instance, the free margin of the valves, which has become suited to its reception by incidental roughness or inequality of surface.

These vegetations cannot, therefore, be regarded as constituting an absolute indication of endocarditis, whose existence requires to be confirmed by the presence of more essential disturbances of texture, but must be considered simply as evidences of a diseased condition of the blood. If, however, such disturbances are present, these vegetations enable us, in the manner already described, to form an opinion in reference to the intensity of the endocarditic process, and the nature of its products.

ABNORMAL CONDITIONS OF THE VALVES, AND ESPECIALLY OF THEIR OSTIA.

§ 1. *Deficient and Excessive Formation.*

We have already considered this subject at p. 146, where we treated of the most important anomalies.

§ 2. *Anomalies of Size,—Hypertrophy and Atrophy of the Valves.*

Anomalies of size in the valves, that is to say, their *superficial enlargement* or *diminution*, usually correspond to an altered thickness of the valves, the former being commonly associated with attenuation, and the latter with thickening of the valves. Exceptions do, however, occasionally present themselves.

Hypertrophy of the valves is found to be almost constantly associated with dilatation of the ostia of the heart, and here we see a healing tendency in nature which endeavours to main-

tain the valves in a state of sufficiency. We observe this in the auriculo-ventricular, as well as the arterial valves, and more especially in those upon the left side of the heart, which, as is well known, is more frequently affected with dilatation of the cavities and ostia. The valves, as we have already remarked, are in these cases usually thin, delicate, and transparent, and so attenuated as occasionally to exhibit actual perforations (atrophy); in like manner the papillary tendons are found to be thinner and more slender in proportion to the extent of the dilatation, while there is a striking thinness and transparency of the whole of the inner lining of the heart.—Exceptions are, however, occasionally observed; the enlarged valve appearing tolerably thick in comparison with the degree of its hypertrophy, which shows that the fibrous tissue of which it is composed must have increased in bulk. This is especially shown in hypertrophied aortic valves by the corresponding enlargement in size and thickness of their nodules, and the fibres passing from them.

If we except the shrivelling of the valves induced by the inflammatory process and its products, *atrophy* of the valves is of rare occurrence, although it may, indeed, very frequently be overlooked. It occurs in diminution (concentric atrophy) of the heart, and is manifested in the form of a shrivelling of the valves, more especially at their free margin, whence the whole valve, including the margin, is found to be thicker and less transparent.—Kingston has observed a case of shortening of the auriculo-ventricular valves, with unaltered thickness, flexibility, and transparency, and with normal width of the annulus, and has described it as a form of *atrophy of the valves*. Shortening may affect one, or more, or all the apices of the valves, and its immediate consequence is insufficiency. It has hitherto only been observed in the auriculo-ventricular valves.

Hypertrophy of the valves affects either their fibrous texture or their investment of endocardium. We have already observed that *hypertrophy* of the fibrous basis of the valves is occasionally associated with their general hypertrophied condition. We moreover frequently notice in the auriculo-ventricular valves, and especially the mitral, both in individuals of advanced age, and in young persons, a pale white, yellowish white streaking of the valve towards its free edge, or a

series of bulgings at the insertions of the papillary tendons, which, however, do not interfere with the function of the valve. No osseous concretions are ever developed in this hypertrophied tissue of the valves. In young persons we occasionally meet with a condition of this portion of the valvular structure, which very probably indicates incipient hypertrophy of the fibrous texture, the free edge appearing swollen, more especially at the insertions of the papillary tendons. This bulging is produced by a pale red, translucent, more or less gelatinous substance, effused into the texture of the valves, from which, as from a blastema, the fibrous tissue is developed. This substance is very commonly found to consist of a translucent, partly homogeneous, and partly indistinctly fibrous mass, in which are imbedded numerous cell-nuclei, and the so-called nucleated fibres. It may be observed in reference to the arterial valves, that hypertrophy of the aortic valves, more especially of their nodules, is not of very rare occurrence. This last-named condition is, however, less frequently observed.

Hypertrophy of the Endocardium is, on the other hand, both more frequent and more intense in the arterial valves, where it more especially affects the aortic valves, as might be expected, from the greater tendency of the left side of the heart and of the trunk of the aorta, to a similar condition of excess of growth in the endocardium and the lining arterial membrane. The valves become thicker in consequence of the deposition of new layers, and the aortic valves more especially at their nodules and free margin present an appearance of bulging; the protuberance being roundish or cylindrical in form, uneven and nodular, and having occasionally a somewhat prismatic or faceted character from the pressure which they mutually exert on one another. The valves thus coalesce with one another, and with the walls of the arteries, by means of prolonged depositions from their lateral insertions. This increase of bulk, which is intrinsically important, is rendered more so in consequence of its secondary effects. A shrivelling process, similar to that by which the arteries are analogously diseased, now affects the valves, which become thicker, full and rigid, and degenerate into a cylindrically formed swelling, and by this means on the one hand contract the ostium, and on the other become insufficient. A bony substance may also be de-

valves in the deposited strata in the form of nodular, round, or dumb-bell-like ossifications, equally important with those affecting the arteries; or, lastly, this deposit may exhibit, (as when it affects the arteries,) an effluviations disintegration and loss of substance resembling an ulcerous process, which, in the same manner as the ossifications, may gradually firm coagula in the form of granular, villous vegetations.

This form of hypertrophy of the valves and its so-called consecutive phenomena, occur only in their greatest intensity in the aortic valves, for the disease invariably exhibits an inferior degree of intensity when it affects the auriculo-ventricular valve on the left side of the heart. This disease is always associated with hypertrophy of the endocardium, and more especially with the deposition of new arterial membrane in the aorta. Although it is most common in advanced life, it does occasionally occur at the age of thirty, or even earlier, and gives rise to the insufficiency of the aortic valves, which is very often gradually and almost imperceptibly developed in persons of advanced life. It is not of endocarditic origin, although it is very often erroneously regarded as a consequence and residuum of endocarditis.²

Atrophy of the Valves.—This disease is manifested by attenuation, unusual delicacy and transparency of the valves, and in its more intense forms by the formation of apertures within them. We have already instanced a condition of attenuation of the valves, as the consequence of their hypertrophied state. We, moreover, observe attenuation of the auriculo-ventricular valves associated with eccentric atrophies and adiposity of the heart. The more highly developed forms of atrophy, in which there are perforations in the valve, are only found in the arterial valves, and more especially in those of the aorta; we do not remember to have observed any case affecting the auriculo-ventricular valves, and we should indeed be disposed to regard this disease as exclusively belonging to the valves of the arteries, if Kingston had not seen a few (three) cases occurring in the former, two of which were in the tricuspid, and the other in the mitral valve. This perforated condition of the valves occurs almost invariably associated

² Dr. Lidd has been led, by clinical observations, to adopt the opinion, that a disease of the valves of the aorta, differing from endocarditis, does actually exist.

with hypertrophy, in consequence of the dilatation of the corresponding ostium. These perforations are almost always situated near the free margin of the valves, and more especially near their insertion, where they originate, increasing in numbers as they spread towards the nodules of the valve. They are at first about the size of a scarcely appreciable pin-hole or of a poppy seed, but after gradually enlarging by the confluence of several into one, they finally attain the size of a grain of millet or a hemp-seed, or even of a pea. When several are present together, they impart a reticular broken appearance to the valve. The perforations are, moreover, surrounded by a smooth margin, and are never round, but oval, elliptical, or fissure-like, and their long axis is at right angles to the free margin of the valve. They are also generally bounded by the fibrous bundles of the valves, so that the atrophy, at least at first, attacks only the thinnest portions. Besides considerable and appreciable attenuation of the valve, and in some cases even perforation, we occasionally find some portions, as, for instance, the free margin, the nodule, and the fibrous bundles passing from it, thickened or hypertrophied.

Perforation of the valves is not of importance unless the apertures are very large, or some among them are deep and seated in the middle of the valve, and cannot be closed by the approximation of the valves; very generally, too, the symptoms are influenced during life by the simultaneous occurrence of heart-disease, as, for instance, dilatation of the left ventricle, and occasionally by the insufficiency of the atrophied valves, that is to say, by their inconsiderable magnitude compared with the dilatation of the ostium. It is, moreover, very probable that attenuated valves, independently of all other conditions, give rise to a change in the sounds of the heart in consequence of diminished resistance, and modifications in the capacity for yielding sounds.

It is in the middle and advanced periods of life, more frequently than in any other, that we meet with atrophy of the valves, in the more intense form associated with perforation; there is thus, in this respect, a perfect harmony with the periods most prone to dilatation of the ostia, of which the aortic opening is the one more frequently diseased. These periods further correspond with the age at which we most

frequently observe excessive depositions of new membrane in the trunk of the aorta, the so-called atheromatous process, and ossification with dilatation; and atrophy of the valves is not uncommonly associated with these diseased conditions of the aorta.

§ 3. *Anomalies of Form.*

Malformations of the valves are alike frequent and varied; but as they are not possessed of any intrinsic importance we do not deem it necessary to enter upon any classification of them. They will be found under their respective sections, and it will therefore suffice to observe, that the malformations affecting the valves in consequence of endocarditis and of hypertrophy of the endocardium, are the most frequent and the most important. The valves of the left side of the heart would appear, from the observations hitherto made, to be the more especial seat of these malformations, more particularly when affecting the endocardium.

§ 4. *Anomalies of Consistence.*

We need do no more than simply refer to the anomalous toughness and hardness of thickened or shrivelled valves, and to the decrease of consistence which accompanies inflammation of the tissue of the valves in the form of relaxation and lacerability, as this subject has already been treated of in a different form under the head of atrophy of the valves.

We would *here*, however, enter more fully into the consideration of a morbid condition of the valves hitherto but little observed, as we know no other place to which we could more appropriately refer the subject, when regarded in a scientific point of view. We allude to a diminution of consistence in the form of an abnormal softness and tendency to laceration of the valves,—an appearance of the greatest practical importance. This disease, when considered in a practical point of view, might be termed a *gelatinous condition of the valve*. The cases in which we have seen it have not been rare, but they were always limited to the valves of the left side of the heart. We find that the valve, either throughout its whole extent, or at individual portions is more yielding, softer, and more readily torn; the faint whitish colour, and the gloss of

the fibrous texture disappear, and are changed to a pale yellowish tinge, approaching here and there to a reddish hue, while the whole becomes translucent. The latter condition probably depends upon the gelatinous non-adhesive substance effused into the tissue of the valve; but yet it is difficult to comprehend how the other anomalies can be produced which we find in this condition of the valve. The tissue of the valve was always found to have disappeared wherever this gelatinous substance was present, and the valve itself, after the removal of this extraneous matter, was observed to be in a state of extreme attenuation or atrophy. The question here arises, is this gelatinous substance a new formation,—a blastema effused here in order to be metamorphosed into fibrous valvular tissue, and for the purpose of strengthening the atrophied valve,—or is it the softened, disintegrated fibrous tissue of the valve itself? The former view appears to us incomparably the more probable, and we are of opinion that this gelatinous substance is the same which, as we have already observed, presents itself in a more dense and tough state, and in the act of undergoing a metamorphosis into tissue, in hypertrophies of the valves. The valve which is rendered soft and lacerable by attenuation becomes still more so from the deposition of this gelatinous substance by which the remaining textural elements are forced asunder. The softness and tendency to laceration of the valve would thus appear to admit of explanation on a mechanical principle, and not on any actual softening process of the tissue.

This gelatinous condition occasionally produces lacerations, more especially of the valves of the aorta. These can be easily distinguished from the perforations already described as produced by atrophy, appearing either as true fringed rents passing lengthways through the valve from its free margin, as fissures in the middle of the valve, or as a laceration or detachment of the valve from its insertional margin.—The gelatinous condition of the valves must, therefore, be classed amongst the more important diseases affecting these structures. The diminished power of tension and resistance must necessarily occasion some modification of the heart's sounds.

This gelatinous condition of the valves undoubtedly admits of cure, since the gelatinous substance may be gradually converted

into a fibrous tissue, and thus condensed, by which means the attenuated valve increases in bulk, and is enabled, if necessary, to enlarge and adapt itself to the size of the dilated ostium.

This condition occurs, at the same periods of life, and under the same circumstances, as atrophy of the valves, either with or without simultaneous hypertrophy.

§ 5. *Separations of Continuity.*

Separations of continuity occur under the forms of laceration of varying depth at any part of the valve, from the margin towards its insertion—as perforation of the valve at different parts more or less remote from the margin—and as a loosening of the valve at the margin of its insertion; and affect the auriculo-ventricular as well as the arterial valves. Commonly only one or other of these forms occurs; occasionally, however, several are present either in one or more of the valves. One very important form of *lesio continui*, which does not affect the valve throughout its whole thickness, but only one of the layers of endocardium and a certain portion of its fibrous tissue, is especially worthy of notice, since it constitutes the basis of *aneurism of the valves*, to which we shall refer more fully in the sequel.

These lacerations of the valves are occasioned by disease of the valvular tissues, arising chiefly from their gelatinous condition; next in frequency, by inflammation (endocarditis); and, lastly, by the loosening of the tissue which accompanies inflammation of the valves. Lacerations of the valves are not only highly important, from the circumstance that their existence presupposes a high degree of the diseases we have already named, but also from their giving rise to valvular insufficiency.

§ 6. *Diseases of Texture.*

To these belong:

a. *Inflammation (endocarditis) of the valves*, which is by far the most frequently observed. This disease is especially important from its results, that is to say, from the morbid changes of the valves to which it gives rise, and the various heart-diseases depending upon the latter alterations.

Endocarditis, as we have already observed, especially affects

the valvular system, which in many cases is *alone* diseased, while in others, it participates in the endocarditis attacking other parts. The valves of the left side of the heart are especially subject to this disease, as we have already seen; and even where the valves on both sides are diseased, those on the right side are always affected in a very much less intense degree.

Inflammation of the valves, in very many cases, is limited to the free margin, whilst, in others, it extends from thence to a various extent towards the insertion of the valve, and not unfrequently attacks the insertion itself, extending to the endocardium of the cavities of the heart and to the tendons of the papillary muscles.

In addition to what has been stated in reference to endocarditis, the following short notice may suffice to explain the characteristics of this disease.

1. *Redness and Injection—Vascularity of the Fibrous Tissue of the Valves*—can only be observed in rare cases of recent endocarditis, for this condition has generally passed into exudation, and cannot be recognised in consequence of the products deposited in the tissue of the valve. Considerable difficulty, moreover, attends the discovery of vascularity, even in recent cases, since it is most frequently masked by the redness of the valves occasioned by imbibition.

2. *Opacity and Bulging of the Valve* are among the most prominent appearances, and depend upon the deposition of inflammatory products in the tissue of the valve. They attain considerable intensity, and are either limited to the free margin of the valve, or extend over a greater portion of it; in some cases the whole valve with its attached margin, or in others with the papillary tendon, is implicated. The endocardium of the valve at several spots loses its usual smoothness and lustre, and the whole has a rough pilous appearance.

3. There may be *Loosening of the Tissue of the Valve*, which, in intense inflammation, predisposes to laceration.

4. *An Inflammatory Product*, which, in addition to the exudation infiltrated into the tissue of the valve and effused and solidified upon its free surface, appears in recent cases as a pilous and granular coagulum in the form of vegetations, or as a membranous exudation having a free finely villous surface, beneath which the valve appears rough, felt-like, and excoriated.

In cases of long standing, these products may often be more readily recognised in the form of a more or less stratified pseudo-membrane, on which depend the thickening and the various forms of adhesions and coalescence of the valves.

5. *Vegetations*, as we have already seen, are deserving of attention, although they cannot be regarded as absolute characteristics of endocarditis.

In the course and as consequences of inflammation of the valves, we observe :

a. *Occasional Laceration of the Valves* in one or other of the above named forms, or laceration of one or more of the papillary tendons; the margins of the rent here generally exhibit an exuberant quantity of vegetations. Laceration is an invariable evidence of the existence of a high degree of the inflammatory process.

b. Inflammation, giving rise to a *purulent product* and to *purulent fusion (suppuration) of the tissue* of the valve, is also not very rare. It may under certain conditions give rise to aneurism of the valves; and is distinguished by an exuberant production of vegetations, which may be considerably diffused, and very frequently undergo purulent disintegration.

c. The most common termination of inflammation of the valves is :

1. *Permanent thickening* of the valve, arising from the product which is deposited in the tissue and on the free surface, and becomes converted into fibroid tissue. The degree of rigidity attained either by the valve and the papillary tendons, or by the former alone, depends upon the extent of the inflammatory process. Contraction of the ostium, and insufficiency of the valve, are frequent results of this condition.

2. This anomaly is rendered more striking when the thickened valve is finally *shrivelled*. This *shrivelling* may occur either in the direction of the perpendicular diameter of the valve, or concentrically with the axis of the ostium. The former produces shortening of the valve, and at the same time insufficiency, and the latter contraction of the contiguous ostium. Both acquire importance in proportion to the extent to which the valve is inflamed, and the contraction is most considerable in the auriculo-ventricular valves, when the inflammation has extended to their margin of insertion.

Hence arise numerous *Malformations of the Valves and of the corresponding Ostia*. Thus the auriculo-ventricular valves, when their free margin and the papillary tendons have been thickened and shortened, present the appearance of a rigid funnel penetrating into the cavity of the ventricle, and exhibiting an elliptic fissure-like opening at the mitral valve and a triangular opening at the tricuspid valve. When the whole or the greater portion of the valve has been thickened and shrivelled, the auriculo-ventricular opening of the left side degenerates into a fissure or button-hole-like aperture surrounded by a rigid string-like ring, while that on the right side appears like a somewhat rounded triangular opening. The arterial valves degenerate into an annular protuberance around the ostium, being of regular height and thickness when the disease is of uniform extent, or irregular in consequence of inequalities in the subjacent surface. In some extreme cases they form a diaphragm inclining with the concavity of its sinus towards the heart and perforated in the centre by a small opening. These are often associated with—

3. *Adhesion, coalescence or fusion* of the different apices of the valve and of the tendons of the papillary muscles to a greater or less extent. These papillary tendons are often found to be fused together into one single or several thick, smoothly roundish rigid strings or bands; but it is only in rare cases that one or more of these apices adhere to the contiguous wall of the heart or of the vessel. It is obvious that such a condition must contribute to produce contraction of the valve on the one hand, and insufficiency of the ostium on the other.

The contractions of the ostia produced by these consecutive anomalies of the valves are, moreover, heightened by the vegetations which so commonly occur.

4. *Osseous concretions* are frequently and variously developed as a secondary disease in the newly formed fibroid tissue of the valves. These are occasionally small, scattered, nodular and roundish, or larger nodular rough band-like formations, and at other times complete osseous rings surrounding the ostium. From these rings the formations diverge in various directions towards the inner part of the valve, passing outwards from the attached margin, where they come in contact with other concretions developed in a simultaneously occurring pericarditic

fibroid exudation. They may also be occasionally connected with concretions in the contiguous wall of the heart, when, together with its endocardium, it had been the seat of inflammation.

These morbid metamorphoses of the valves are, as we have already observed, by far the most frequent originating causes of dilatations and hypertrophies of the heart. Inflammation of the valves and its results must not be confounded with hypertrophy of the valves,—with excessive endocarditic deposition on them and its metamorphoses.

By way of supplement to this subject we will now treat of the so-called *Aneurism of the Valves*.

Aneurism of the Valves.—Some writers (Thurnam) have applied this designation to a morbid condition of the valves, which has indeed some affinity with aneurism, more especially if we adopt Scarpa's theory of *spurious Aneurism*, (the *mixed Aneurism* of others.)

From our own observations, we should be led to divide aneurism of the valves into two forms, and to compare them with the two forms of aneurism of the heart we have already described, although they do not indeed strictly correspond to one another, since only *one* (namely the *acute*) form of the disease in the valves corresponds with acute aneurism of the wall of the heart, while we have observed *no form of aneurism of the valves*, corresponding to *chronic* aneurism of the heart. We would, however, include under this head consolidated (cured) aneurism of the acute form, affecting a valve, whose continuity is still undestroyed, although it may present unimportant sinuosities, such as are sometimes observed at the mitral valve near the free edge. We have found these structures in only one case, together with the residua of endocarditis, at the aortic valves, and, if they were not consolidated aneurisms of the acute form, they must have been produced by a hernia of one of the lamellæ of endocardium, through the fibrous layer of the valve. —This class would necessarily include the three cases described by Thurnam (one of which affected the mitral, one the tricuspid, and the third one of the aortic valves), since he regards them as dependent on gradual extension of the valves.

The following is the *mode of origin of acute aneurism of the valves*, according to the observations made by ourselves and

others. In the course of intense inflammation of the valves, a *læsio continui* is produced in the valve, affecting only one of the laminae of endocardium, and a layer of the contiguous fibrous tissue.

1. This *læsio continui* appears either as a separation or fissure of the structure, and may occur,—

a. As the consequence of the condition of loosening and lacerability induced by inflammation,—

b. Or it may arise from the loosened condition of the tissue, observed in the neighbourhood of an abscess, in the parenchyma of the valve.

2. A *læsio continui* may also be the result of an abscess proceeding from the lowest part of the valve, and penetrating towards or even through its endocardium, or in other words it may be owing to a final suppuration of the endocardium. (In one specimen in our collection, a sinus even passes from an abscess in the substance of the heart towards the aortic portion of the mitral valve; above this the valve is torn from the ventricle, and the whole sinus thus converted into an aneurismal sac.) It is however very questionable whether the endocardium is actually in a state of suppuration in such cases, or whether it may not rather be lacerated above the adjoining abscess, in which case the whole process would essentially belong to that which has been considered under *b.*

When there is a tendency to laceration of the valve, this will occur with a frequency proportional to the extension of the aneurism in the direction towards which the blood flows to the valve,—the auriculo-ventricular valves being lacerated in the direction of the auricle and the arterial valves in that of the ventricle,—and especially when the *læsio continui* affects the surface of the valve against which the blood is propelled.

Thus where the valve has been perforated to a greater or less extent, the blood which impinges on it, penetrates into its parenchyma, and causes more or less extensive infiltration. By this means the yet uninjured portion of the valve assumes the appearance of a projecting tumour on the corresponding surface; and becoming, as it were, inflated, constitutes *valvular aneurism*, in the form in which we have observed it, and to which the following remarks apply.

This tumour is usually about the size of a pea or a bean,

although, after continued attenuation of the layer of the valvular tissue of which it consists, it becomes as large as a hazelnut, or even a pigeon's egg. The tumour is especially capable of such an enlargement at the auriculo-ventricular valves, in consequence of the more abundant mass of the fibrous tissue occurring in them.

Its *form* is round and hemispherical, or frequently so far irregular that it presents various sinuosities in the circumference of its base, as well as in its arched portion. It generally extends over a considerable space, in consequence of the widely diffused infiltration of the blood into the parenchyma of the valve.

Its *aperture* although originally a fissure-like rent, is generally round, and has fringed margins, which, together with the circumference of the valve, are covered with luxuriant vegetations.

Its *cavity* is filled with a variously discoloured bluish red, reddish grey, yellow-reddish, dirty white, solid, or more frequently loose, soft coagulum, which very often becomes disintegrated like the globular vegetations.

Aneurism of the valves is, therefore, as may be seen from what has been already stated, an *acute formation occasioned by a considerable degree of inflammation of the valves*.

These tumours in general *terminate* somewhat speedily in lacerations. This usually occurs in the more intense aneurisms of the auriculo-ventricular valve at the highest point of the aneurism, or at the summit of one of its various pouches, in the form of a small fissure-like rent, inclining from its circumference towards the opening, and having its margins speedily covered with vegetations.

Death does not follow from this mode of termination of the disease, but results from the endocarditic process and the corresponding disease, to which the latter gives rise in the blood.—In the rare cases in which this aneurism is consolidated, (that is to say, where it has its opening and the walls of the cavity covered with membrane,) it constitutes a chronic aneurism, and, like simple or hernial sinuosities, continues longer (see the cases recorded by Thurnam and others), and may become fatal through consecutive diseases in connection with other co-existing heart-affections. It may be observed, in reference to the size of such aneurismal pouches of the valves,

that in one case seen by Thurnam the tumour had attained the unusual dimensions of a large walnut.

Our own experience coincides with that of most foreign observers, in having discovered this aneurism on the valves of the left side only,—a circumstance that corresponds with the relation of endocarditis to the same side of the heart. It is probably always more extensive at the auriculo-ventricular valve, on account of the greater development of the parenchyma in that structure whilst it is lacerated in the arterial valves soon after its formation, and may thus terminate in a large fissure.—Thurnam, as has been already observed, found aneurism of the tricuspid valve in a heart in which there was a communication between both ventricles. In this case there were four aneurismal pouches on the valve.

From the above observations it will easily be seen, that although aneurism of the valves possesses a scientific interest, it is not of much practical importance when considered either on its own account or in reference to the intense disease from which it arises.

b. Adventitious Structures.—These are almost entirely limited to the occurrence of fibroid tissue and anomalous osseous substance, (ossification,) both of which are of very frequent occurrence.

1. The *fibroid tissue* presents various anomalies in reference to the elements of which it is composed. Thus, for instance, as will be seen under their respective heads:

a. It is found to be abnormally developed in hypertrophy of the valves.

b. It occurs in excess in those products (exudations) of inflammation of the valve which are developed in the tissue as well as on its surface.

c. The endocardium, deposited in excess on the valve, usually undergoes some metamorphosis of this nature.

d. A similar metamorphic process is observed in reference to the vegetations of the valves.

2. *Osseous formation* occurs in various essentially different forms, to which little attention has hitherto been paid:

a. The fibroid tissue produced by the process of inflammation, occurs in the above mentioned form of protuberant, roundish, and band-like concretions. They are originally de-

veloped in the inner part of the thickened and shrivelled valve, from whence they increase in circumference, owing to the continued ossification of the fibroid tissue, and at length come to view uncovered in different portions of the cavity of the heart. They are closely analogous to the ossifications of fibroid exudations found in serous membranes.

b. The endocardium abnormally deposited upon the valve becomes ossified. These concretions are very frequent at the aortic valves, and of rarer occurrence at the mitral valve. In the former case, they frequently attain considerable bulk; but, in the latter, they are merely small plates. They correspond with the ossifications of the inner lining membrane which is deposited in excess in the arteries, and are originally developed, like these, in the lowest and earliest strata, being denuded and coming to view when all have been ossified. Such are the ossifications frequently observed in advanced life, which have no connection with pre-existing endocarditis, however they may be associated with endocarditic products.

c. In addition to these concretions there is a third variety,¹ which is highly interesting from the many analogies with which it is associated. It presents itself most frequently (more especially at the aortic valves) as an osseous concretion in a stalactitic form, or as a rough granular agglomeration. These calcareous formations constitute a metamorphosis or conversion of the vegetations on the valve into bony and chalky matter. As might be expected, and in accordance with experience, they are frequently found to be associated with one or more of the two above named forms, (more especially, however, with the ossifications considered under *a.*) which are developed in the valve after it has been thickened by inflammation. These stalactitic osseous masses occasion and promote the continued formation of new vegetations, and are consequently very commonly surrounded by them.

Even the normal tissue of the valve becomes of a dirty yellow, faded colour in advanced life, and exhibits a layer of fat and calcareous salts in a finely comminuted form.

Besides these secondary processes, we will consider:

3. The *atheromatous disintegration* of newly deposited endocardium as it commonly occurs in a low degree on the valves.

¹ Oesterr. Jahr. B. xxiv, St. I.

4. Finally, in rare cases, where the necessary constitutional conditions are present, the vegetations on the valves of the heart exhibit a *carcinomatous* character, the cancer being usually of the *medullary* kind.

Review of the Anomalies of the Valves, and more especially those producing Contraction of the Ostia and Insufficiency.

We have endeavoured, in the foregoing observations, to indicate those cases in which an anomaly of the valve produces contraction of the corresponding ostium, or the causes by which the valve itself becomes insufficient.

The causes on which *contraction* of an ostium depends, the mode in which it is variously developed through thickening or rigidity of the valvular apparatus, vegetations, &c., and the manner in which it may finally give rise to consecutive heart-diseases, in the form of hypertrophy and dilatation, all alike self-evident.—This contraction is frequently so considerable, that the diameter of the auriculo-ventricular opening, more especially on the left side, scarcely equals that of the nail of the little finger, or even of a goosequill, while the arterial openings would not admit of the passage of anything larger than a crowquill.

The condition of the valves known as *insufficiency*, has only been adequately considered by modern observers. By the term *insufficiency*, we understand that condition of the valves in which they are unable to close the ostium, and thus allow the blood to return or regurgitate into a cavity of the heart which would be isolated if the ostium were completely closed. In this way, the insufficiency of the auriculo-ventricular valves allows a portion of the blood to return from the ventricles into the auricles during the systole of the former, while the insufficiency of the arterial valves allows the blood to return into the ventricle during its diastole.

As might be expected, we frequently find that one and the same anomaly of the valves produces contraction of the ostium and insufficiency. The latter is especially owing to the following anomalies.

1. *A relative diminution in the size of the Valves with dilatation of the Ostia*, the degree of the former depending on the intensity of the latter. As we have observed, the valves

in these cases are commonly enlarged at the expense of their thickness and power of resistance, and they may continue to remain sufficient when the ostia are very considerably dilated.

2. *Perforation of the Valve*, in consequence of atrophy. It must be very well marked before it can give rise to any considerable degree of insufficiency.

3. *Laceration of the Valve*, under various forms, in consequence of the gelatinous condition of the valve, or, perhaps, still more from its inflammatory state. The degree of insufficiency is increased in proportion to the extent of the laceration.

In like manner, *laceration* of one or more of the papillary tendons produces *insufficiency* of the valves.

4. *Shrivelling and Shortening of the Valve and its Tendons*;—the valve does not close the ostium, in consequence of its rigidity or its insufficient length. This insufficiency in the case of the mitral valve is in general owing to well-marked endocarditis; in the aortic valves it is often very slowly developed, and, in advanced life, it is generally owing to an excessive deposit on the endocardium. This is the most frequent form of insufficiency, and the one which attains the most considerable degree of intensity; it is usually attended with contraction of the ostium, owing to the rigid, thickened, and shrivelled state of the valve. It will be seen, from what we have already stated at p. 226, that it is only in very rare cases that insufficiency ensues in consequence of shortening the valve depending on atrophy.

5. *Fusion of the Valves with one another, or their Coalescence with the wall of the Heart or Vessel*, generally induces a high degree of insufficiency in combination with the above named conditions.

It will be easily understood, that not only carditis and its metamorphoses, but also fatty degeneration of the muscular substance of the heart, especially when seated in the papillary muscles, may induce insufficiency of the valves.

Insufficiency of the valves gives rise to the same heart-diseases as contraction of the ostia; but, as has been already observed in p. 162, it has not been clearly demonstrated whether it specially induces dilatation, and on the other hand whether the stenosis specially gives rise to hypertrophy.

SUPPLEMENT.

Cyanosis has so long constituted a special subject of anatomical inquiry, that our work would be incomplete were we to omit stating our views in reference to this affection, and the relation it bears to heart-diseases. We must however observe, that our opinions are not derived from a careful consideration of all the known cases of cyanosis of the heart, but are, properly speaking, the mere expression of the views we have adopted from personal observation, and from the study of a limited number of the cases reported by others. (Morgagni, Ferrus, Louis, &c.)

A distinction is commonly made between cyanosis, arising from organic *heart-disease*, acquired in advanced periods of life, or from *diseases of the lungs*, and cyanosis depending upon *congenital malformations of the heart*. The latter form is specially designated *cardiac cyanosis*; but we shall see in the sequel that both forms are identical in origin and character.

The cause of cyanosis, when depending upon original malformation of the heart, has usually been sought in the admixture of the venous with the arterial blood, either in the ventricles, the auricles, or the trunks of the vessels; and this admixture, together with the cyanosis, has been supposed to arise from a deficiency in the septa between the cavities of the heart.

According to our view, cyanosis does not arise from an admixture of the venous and arterial blood, which is in many cases very problematical, and not unfrequently altogether impossible, but depends rather upon *an obstruction in the passage of the venous blood into the heart, and upon an overcharging of the venous system, which is either transient or habitual, according to the circumstances of the case, and induces a corresponding repletion of the capillaries*. We moreover consider that all cyanoses generally admit of being classed under one head, however the causes from which they immediately arise may differ in depending on original and congenital, or acquired anomalies of the heart and lungs.

We are led to conjecture from our own experience, con-

firmed by the observations of others, that cyanosis never arises from malformations of the heart, consisting in deficiency of the septa, unless there exists at the same time some special anomaly of the arterial trunks, as narrowness or insufficiency of calibre, or contraction of the ostia of the heart. We will limit ourselves in the following notice to the most remarkable forms of this affection, and to cases which admit of being observed during a prolonged period after birth.

Patency of the Foramen ovale, although not uncommonly observed after death, is very generally not manifested by any symptoms during life, unless it occur in connection with some anomaly of the arterial trunks. This circumstance is the less remarkable when we remember that, under similar conditions, there may be an entire absence of the auricular septum, unaccompanied by the presence of cyanosis.

This patency cannot, in ordinary cases, be referred to any definite cause, and is, as far as we know, purely accidental; but, in some comparatively rare instances, it certainly depends upon an anomaly of the arterial trunks, the patency of the ductus arteriosus, the presence of apertures in the ventricular septum, endocarditic metamorphosis of the valves, giving rise to contraction of the ostia in the fetus, or upon pulmonary diseases, as catarrh, atelectasis, &c.

It must be observed, in reference to the question of a mixture of the venous and arterial blood, in patency of the foramen ovale, that in ordinary cases it is most probable that no such admixture actually occurs, inasmuch as the masses of the blood accumulated in the auricles equipoise one another, and the valve is pressed against the septum by the blood in the left auricle.

Symptoms of cyanosis do not occur even in cases of considerable deficiency of the valve of the Foramen ovale, without or even with the persistence of the foetal condition of the Eustachian valve, which carries a portion of the blood of the Vena cava to the Foramen ovale, although in the latter case there is necessarily an admixture of venous and arterial blood.

In those cases, however, in which the patency exists conjointly with or is dependent upon the above named anomalies, the symptoms of cyanosis are necessarily present, although this mixture of both kinds of blood is not invariably effected,

as is commonly assumed, by the afflux of venous to arterial blood. The mode of admixture depends upon the nature of the accompanying anomaly in the vessels or heart. If for instance there is abnormal narrowness or obstruction of the pulmonary artery, the blood of the right auricle will be mixed with that of the left auricle in consequence of the obstruction to the escape of the blood from the right ventricle of the heart; but if, on the other hand, the aorta be the seat of the anomaly in question, the arterial will be carried to the venous blood. Either of these conditions will be induced in alterations of the ostia, occasioned by foetal endocarditis, according as the right or the left side of the heart has been the seat of this process.

The patency of the Ductus arteriosus involves patency of the Foramen ovale from the right auricle, although not always in the manner usually assumed. It is supposed that the quantity of the blood in the left auricle diminishes with the width of the latter, as it flows into the aorta, and that a continued current of blood from the right auricle prevents the closure of the Foramen ovale. There are cases, however, in which the form of the open Ductus Botelli and its two mouths, as, for instance, its expansion from the direction of the aorta, render it highly probable that the blood flows from the aorta towards the pulmonary artery, and, in such cases, the transmission of the blood of the right auricle, and the patency of the Foramen ovale, are the result of the excessive fulness of the former, arising from the passage of the aortic blood into the pulmonary artery, and the consequent obstruction to a free discharge of blood from the right side of the heart. In either case, whether the venous blood passes into the arterial, or the arterial blood into the venous, the presence of cyanosis will occasionally be manifested, in consequence of the inability of the blood in the Venæ cavæ to pass into the diseased heart when already in a state of dilatation.

Very considerable deficiency, or even the entire absence of the auricular septum, although necessarily accompanied with the admixture of the venous and arterial blood, does not give rise to cyanosis where the arterial trunks are normal. Numerous observations testify, however, that this deficiency very probably seldom exists unaccompanied by an anomaly of

the vascular trunks, although its presence may frequently be overlooked. This consists in an *obvious* narrowness of the trunk of the aorta, which gives rise to a remarkable degree of cyanosis, although *the arterial blood abundantly passes into the venous*. Narrowness of the trunk of the aorta, like contraction of the aortic opening, occasions active dilatation of the left ventricle, extending to the left auricle, and lastly, to the right side of the heart, through the capillary system of the lungs. The immediate consequence of the obstruction to the passage of the blood from the left ventricle and the auricle, occasioned by the narrowness of the trunk of the aorta, is undoubtedly to carry a portion of the arterial blood of the left towards the right auricle. A more remote consequence of the obstruction to the discharge of the blood from the left side of the heart, is to impede the passage of the blood of the Venæ cavæ into the right side of the heart, and we then have cyanosis as the result of the overloading of the capillaries from the Venæ cavæ.

It is evident that in these cases there will generally be a considerable degree of active dilatation of the right ventricle, especially of the Conus arteriosus and the trunk of the pulmonary artery. Bouillaud is unable to explain this circumstance, otherwise than by assuming that the right ventricle becomes arterialised from contact with the arterial blood, which enters it from the left auricle.

It would appear, from numerous observations, that a *deficiency of the ventricular septum*—as its perforation—and the communication consequently established between the two ventricles do not, in all probability, give rise to cyanosis, unless there exists a simultaneous anomaly of the arterial trunks. For, in the absence of this predisposing cause, and only under certain conditions, such as mental emotion, bodily exertion, or disease of the lungs, cyanotic symptoms are of rare occurrence and of a transient character. It must, however, be observed, that important anomalies of the vascular trunks are of such common occurrence with deficiency of the ventricular septum, that the latter condition is almost constantly associated with *excessive cyanosis*.

The anomalies of the vascular trunks most commonly associated with absence of the ventricular septum, are a more

or less striking narrowness and obstruction, or even the complete closure of one or other of the arterial trunks, more especially the pulmonary artery, so that the aorta springing from both ventricles supplies the circulating system generally, and the lesser circulation especially, by means of anomalous pulmonary branches. The aorta here shows itself inadequate to the discharge of the blood from both ventricles, and the cyanosis must, therefore, undoubtedly arise from the obstruction opposed to the entrance of blood from the venous system, for we find in numerous cases of deficiency of the septum, where the vascular trunks are normal as well as where they are displaced, that cyanosis is either wholly absent, or that it occurs only on certain occasions, as, for instance, in pulmonary disease; that is to say, it arises in consequence of the retention of the blood in the venous system, by which the passage of the blood from the right side of the heart to the lungs is impeded; there can, however, be no doubt that an admixture of the venous and arterial blood is constantly taking place.

In like manner, where the entire ventricle is not properly developed, it and the vascular trunks to which it gives rise are rendered insufficient for the discharge of the mass of the blood.

The heart, in all these cases, exhibits dilatation and hypertrophy, which either affect both ventricles uniformly, or one more than the other, especially the right one, so that the heart retains its foetal character, more especially in reference to the mutual relation of the ventricles.

Cyanosis is either continuous, although commonly remittent, or results from certain definite causes, among which we may reckon all those which influence the free passage of the blood through the lungs and heart,—as mental emotions, violent bodily exercise, &c. Pulmonary diseases may, perhaps, be regarded as the most powerful of any; and among these, the pulmonary catarrh which affects children and young persons is more especially influential in giving rise to symptoms of cyanosis; the more so, perhaps, because habitual bronchial catarrh is very commonly found to be associated with the above named malformations, in consequence of the insufficient emptying of the pulmonary vessels into the heart. Cyanosis occasionally appears in childhood and puberty, when it is undoubtedly to be ascribed to a want of relation originating at

this period of life, between one or other of the arterial trunks and the heart.

The appearances presented after death correspond with the character of the cyanosis, whether it be constant or transitory, and whether it have arisen from different known and obvious causes, or be owing to influences either unknown or unexplained; and we thus find that some persons suffering from cyanosis manifest retarded development, deficient nutrition and animal heat, and general debility, and die prematurely, while others exhibit merely a very slight depression of the functions of organic life. In some cases, in which the heart presented conditions admitting of the admixture of venous and arterial blood, all the functions were fully performed;—a circumstance that it has been attempted to explain by the assumption that no admixture of the two kinds of blood occurs, in consequence of the equal development of both sides of the heart.

A morbid form of growth frequently associated with cardiac cyanosis, is the drumstick-like or club-shaped form of the ends of the fingers, with a corresponding convexity of the nails. This phenomenon has not been explained, and if, as has been asserted by different observers, a similar malformation is acquired in pulmonary phthisis, it may serve, from its association with pulmonary cyanosis, to confirm our view of the mode of origin of cardiac cyanosis.

An important observation militating against the ordinary view of the mode of origin of cyanosis has been made by Breschet, who found, in one case, that the subclavian artery of the left side sprung from the pulmonary artery, while the extremities presented no anomalous colour. We find, however, that there exists a species of local cyanosis in those cases in which the return of the venous blood has been obstructed by the afflux of arterial blood into a vein, as in varicose aneurysm. Finally, in the fœtus there is no cyanotic colour, although there is a constant admixture of the arterial and venous blood. (Fouquier.)

Capillary hæmorrhages of the most various organs constitute phenomena in every way important in cardiac cyanosis. They most commonly occur as bleedings from the lungs, and are undoubtedly occasioned by the rupture of the overcharged capillaries. They afford as strong a confirmation of our views

as a case which fell under our notice of a cyanotic boy, aged 8 years, who died from laceration of the trunk of an insufficient aorta beyond its arch, and in whom there was an opening in the ventricular septum, closure of the pulmonary artery, and an origin of the aorta from both ventricles.

Cyanosis, or the abnormal formation of the heart on which it depends, may terminate in death, either suddenly and rapidly, or slowly, in the same manner as in acquired heart-diseases.

There is an anomalous form of cyanosis depending on *original narrowness of the arterial trunks, associated with a normal formation of the heart*, which constitutes a transition stage from the cyanosis, arising from malformations of the heart and of the arterial trunks to that form of cyanosis which is a symptom of acquired heart-disease. This anomalous narrowness, associated with a normal formation of the heart, extends in various degrees to the aortic trunk; and, like many other phenomena of cachexia, occurs most commonly during childhood and in puberty.

Finally, *cyanosis is a common symptom of many heart-diseases*, such as dilatations and hypertrophies of a higher degree, together with the anomalies of the valves, from which they originate. These diseases also commonly give rise to acquired anomalies of the vascular trunks, such as contraction and obliteration, acquired communications of the aorta with the pulmonary artery and with the Venæ cavæ, and the consequent entrance of arterial blood into the two last named vessels. These forms of cyanosis very frequently do not appear decidedly until advanced periods of life, although the heart-disease may have been acquired in early childhood, if it be not even congenital. It is still problematical whether this form of cardiac cyanosis can be acquired by *the re-opening of the closed Foramen ovale and by a morbid perforation of the septum, owing either to inflammation and suppuration, or to a fissure*. This view of the possibility of the re-opening of the Foramen ovale originated at a period when too much importance was attached to its patency, and would appear to be especially designed to serve for the completion of the whole theory. The cases recorded of acquired morbid perforation of the septa are certainly not wholly improbable, but the previous history of these cases affords us no convincing grounds for the assumption

of the process of inflammation and suppuration or the existence of a fissure. These cases are also incapable of solving the questions whether these processes may not date from the period of foetal life, and whether, therefore, the morbid perforation may not be a congenital heart-disease; nor do they show whether the case may not be one in which the products and residua of inflammation belonged to a process subsequent to the perforation rather than to one by which this process was effected. This perforation deserves the more consideration, since traces of old or recent endocarditis have not unfrequently been found in hearts presenting such anomalies of formation.

Cyanosis may not only be derived from the heart (where it most commonly originates in the right side), but it may also depend on the most various congenital and acquired *diseases of the lungs*, which impede the circulation of the blood in the capillaries. The insufficiency of the pulmonary capillaries to carry the blood from the right side of the heart, causes an impediment in the discharge of the venous system into the right cavities of the heart, and thus gives rise to cyanosis; and it, moreover, as is clearly observed when of long continuance, induces active dilatation of the right side of the heart, the intensity of which corresponds to the degree of the impermeability of the pulmonary capillaries. We purpose in a future page to treat this subject more in detail.

Diseases of the left side of the heart, such as dilatation and hypertrophy of the ventricle, but more particularly contraction of the auriculo-ventricular opening, occasion the right side of the heart to be over-filled, in consequence of the obstruction opposed to the discharge of the blood from the capillaries of the lungs; and hence we have the phenomena of cyanosis, usually with extension of disease (dilatation and hypertrophy) to the right side of the heart.

Cyanotic phenomena of various degrees of intensity may also depend on conditions of excessive density, and on continued compression of the lung, (as for instance from exudations), on atelectasis of the lung, on catarrh and bronchial dilatation, emphysema of the lung, extensive pneumonia and pneumonic induration, pulmonary tuberculosis, &c., in like manner as on narrowness and closing of the arterial trunk, and admit of an equally easy mode of explanation. These

phenomena of cyanosis may, moreover, either when congenital or acquired soon after birth, obstruct or wholly prevent the involution (closure) of the fœtal passages.

All cyanoses, or rather all forms of disease of the heart, vessels, or lungs, inducing cyanosis of various kinds and degrees, are incompatible with tuberculosis, against which cyanosis offers a complete protection, and herein we find a key for the solution of the immunity against tuberculosis afforded by many conditions which at first sight appear to differ so widely from one another.

III.—ABNORMAL CONDITIONS OF THE ARTERIES.

§ 1. *Deficiency and Excess of Formation.*

We have already treated, in their connection with anomalies of the heart, of all anomalies or other defects of structure of the two arterial trunks, in so far as they present any true pathological interest. We would here only notice the following facts.

It is extremely doubtful whether there is a complete *absence* of a vascular system even in the most imperfect monsters, although extreme deficiency and a very imperfect indication of the two systems, is common in these cases. Where there is a deficiency of individual portions of the body, there naturally exists some anomaly in the corresponding portions of the vascular system, which will present deficiencies corresponding in intensity to the degree of arrest of development.

Supernumerary parts present a corresponding *excess of formation* in the vessels, although it must be observed that as a multiplication of organs is not uncommonly merely apparent, the multiplication of the arteries and veins implicated in the anomaly is only apparent, indicating the mere cleavage or subdivision of a trunk. We have already spoken, and purpose treating more fully, of the excess of vessels supplying different new formations. To these *anomalies in the number* of the individual vessels belong :

§ 2. *Anomalies in the Origin, Course, &c., of the Arteries.*

These, which are known as varieties, are very numerous, and are in part produced by anomalies of position and form in the corresponding organs. Several are highly important in

reference to operative surgery; but as we are unable, from personal experience, to add any new facts in relation to this subject, we would refer our readers to the Monographs in which it has been treated, and to the ordinary Systems of Surgery.

§ 3. *Anomalies and Diseases of Texture.*

We commence with the consideration of these anomalies, because they are both intrinsically important, and constitute the foundation of the principal consecutive anomalies and diseases, and because a knowledge of them is absolutely necessary for the right comprehension of the great majority and the most important of the alterations affecting the calibre and thickness of the walls of the arteries, their separations of continuity, and the subsequent phenomena to which they give rise. We shall institute various comparisons with the corresponding textural diseases of the veins, and we would here specially refer, on all these points, to the remarks we shall have to make subsequently on the diseases of the veins.

a. Inflammation of the Arteries—Arteritis.—In the first place it will be necessary clearly to comprehend whether there actually exists a spontaneous *arteritis*, and whether that special form of *arteritis* ever occurs which is commonly supposed to be met with in our hospitals and dead-houses.

If under the term *arteritis* we understand an acute inflammation, in which the inflammatory products are deposited or exude, as in *phlebitis*, (inflammation of the serous membranes, &c.,) on the free surface of the lining membrane of the vessel—that form of inflammation which is supposed to give rise to those appearances of redness commonly observed after death in the inner coat of the arteries—it will be necessary to notice the following points in reference to this subject:

1. The absence of vessels in the (yellow) circular fibrous coat, and more especially in the inner coat of the vessels, forbids our assuming the possibility of inflammation in these layers. This is also fully confirmed by experience, and we find that the redness observed in these coats, more especially in the lining membrane of the vessels, is obviously owing to imbibition, which is developed after death, and possibly even during life, with a rapidity proportional to the state of decomposition of

the blood. The coloration always proceeds from the inner surface of the vessel without a trace of vascularity, and penetrates to different depths in the yellow membrane; whilst there is no product of inflammation to be discovered either on the inner surface of the vessel or in the tissue of either of these arterial coats. The phenomena manifested during life by the supposed arteritis, are in such cases always dependent on primary or secondary disease of the blood.

2. The cellular sheath of the vessel is alone capable of inflammation, and we are here led to inquire, whether this inflammation can deposit its products on the inner surface of the artery, under what conditions this may be done, and, what experience teaches us in reference to this subject.

The circular fibrous coat in the larger arteries, as, for instance, in the trunk of the aorta, exhibits so great a thickness, together with such density of texture, that we are unable to comprehend how it can be permeated by an exudation, unless by the agency of an acute process. This is fully confirmed by experience; for, at all events, we have never detected any such process in the trunk of the aorta, or, in other words, never observed arteritis in the sense in question; and we are, therefore, led to deny, or at all events to doubt the correctness of the observations recorded in reference to this subject. According to our view, the supposed pseudo-membranes on the inner surface of the aorta, and its redness, together with the fibrinous plug obstructing its canal, are separated and coagulated from the diseased blood.

We, moreover, regard it as very doubtful whether pus is ever produced in the inner coat of the arteries or between this and the middle one, nor do we attach any great weight to the observations of Andral, in which, as he asserts, he found some half dozen abscesses of the size of hazel nuts under the inner coat of the aorta.

Experience alone is able satisfactorily to determine the limits at which the thickness and density of the texture of the circular fibrous coat cease to oppose an absolute obstacle to the imbibition and permeation of an exudation produced in the cellular sheath of the artery, or to its appearance on the inner surface of the vessel. We find, indeed, from observation, that such inflammation frequently exists in the *femoral arteries*, espe-

cially in women, and likewise in the *umbilical arteries of newborn infants*; and we are hence led to conclude, that it may occur in all those arteries generally which possess a like organisation.

The *anatomical appearances of acute arteritis*, considered within the limits we have assigned to its occurrence, are:

1. *Injection, Redness of the cellular sheath of the vessel.*—This is commonly no longer distinctly apparent after the establishment of those products which we are about to name.

2. *Infiltration of the cellular sheath*, with a serous, sero-fibrinous, partially solidifying moisture, causing puffiness; in some few cases the tissue exhibits purulent exudations, which are either diffused, or limited to individual points, or grouped into circumscribed foci.

3. *Extreme lacerability*—the facility with which the cellular sheath may be removed from the circular fibrous coat.

Hitherto we have only enumerated the appearances attending inflammation of the cellular tissue. (See vol. III, p. 4.)

4. The *circular fibrous coat* appears loosened and succulent; admits readily of being drawn into fibres and separated from the elastic coat; is commonly blanched, and sometimes coloured in different shades of red from the inner surface of the vessel through imbibition, although without any apparent injection. The *lining membrane* of the vessel is loosened, and may easily be detached or torn; its free surface is dull, and occasionally exhibits a felt-like or wrinkled appearance; it is either pale or reddened through imbibition.

5. The vessel is frequently, although not always perceptibly *dilated*, which is owing to the paralysis of the elastic layer of the cellular sheath and the circular fibrous coat. A coagulum of blood acting as a plug is lodged in the canal of the vessel, whose bore is thus more or less completely filled and obstructed.

6. The *presence of a free product (exudation) covering the inner surface of the vessel in the form of a pseudo-membrane* is, in most cases, problematical. It occasionally occurs in the form of a soft, pale yellowish, or yellow-reddish layer, differing from the inner lining membrane of the vessel, as well as from the peripheral stratum of the plug. In more frequent cases, that which may be regarded as a free product, is merely the

outer layer of the plug in the act of being metamorphosed into a structure analogous to the inner coat of the vessel. In these cases, the exudation thrown out on the inner surface of the vessel is taken up into the blood before it has been consolidated and has thus given rise to the formation of an obstructing coagulum. In many cases a portion of the exudation is solidified under one of the strata forming the inner coat of the vessel—below the epithelium and the longitudinal fibrous coat—and these strata being thus loosened are thrown off. They form a covering to the plug which projects into the canal of the vessel.

These appearances constitute the so-called *adhesive arteritis*.

7. In rare cases arteritis gives rise to a partially or wholly *purulent exudation*, which may be recognised by the following appearances :

a. The inner surface of the vessel sometimes distinctly exhibits a thin layer of purulent exudation, which is partially attached to the plug.

b. This exudation, which is discoloured and loosened, undergoes a process of softening, both in its interior and at different points of its circumference, and is reduced into a puriform semi-fluid mass, or into a fluid exhibiting a corresponding degree of decoloration.

c. The inner and the circular fibrous coats are swollen, unusually succulent, of a somewhat yellowish colour, loosened and stratified, and are distinctly infiltrated and permeated with the purulent exudation.

d. The whole of the lining membrane of the umbilical arteries of new-born infants is frequently found to be detached from the yellow membrane, which again is separated from the elastic coat by means of the exudation which is produced from the cellular sheath, and is, for the most part, accumulated between these coats.

e. The cellular sheath exhibits the above named signs of inflammation in a highly developed form, having purulent exudation diffused over the tissue or accumulated in circumscribed foci.

This form of arteritis deposits, therefore, as appears from the above observations, a product which is either capable of coagulation and solidification, and of being metamorphosed

into tissue, or is of a purulent character. The first of these forms is commonly termed *adhesive*, on account of the obliteration of the vessel to which it very usually gives rise; it is of much greater frequency than the other form.

In reference to the modes of *termination* and the metamorphoses of the products of this form, we must notice the following particulars:

1. Adhesive arteritis occasionally passes into *resolution*, as does more frequently adhesive phlebitis; the products of inflammation in the tissue of the coats of the artery are resorbed, while the plug is gradually dissolved, and taken up into the blood in a finely comminuted condition.

2. *The ordinary termination is permanent and more or less complete obliteration, which, in its turn, gives rise to atrophy of the vessel.*—The plug in the vessel gradually shrivels, being decolorised and converted into a fibroid string. When the plug has entirely filled the vessel, and is attached to the inner wall by means of one of the above named structures, the coats of the vessel will be found to adapt themselves to its shrivelling, while the vessel closes around it into a solid cylinder. But where the plug has not completely filled the artery, and is only attached at some points to the wall of the vessel, or where the shrivelling and metamorphosis have been effected too rapidly to allow of the walls of the artery following the process uniformly, and the adhesions have therefore been drawn aside and partially loosened by the flow of blood, the obliteration will be incomplete. In these cases the artery is occupied by a fibroid cord or string, which closely adheres at certain points to the wall of the vessel, although free at all other parts, or is, at the same time, attached in different places by means of string-like structures or pseudo-membranous plates, so that the calibre is very much contracted, and the circulation, more especially in the smaller vessels, correspondingly impeded. In consequence of complete closure, the specific tissue of the artery, more especially the circular fibrous coat, very rapidly disappears, and the vessel becomes converted into a hard fibroid cord enveloped in cellular tissue.

Bony substance may be developed in this fibroid cord, which may be ossified over various extents of its surface.

It is with purulent exudation leads, in some few

cases, to complete or partial *suppuration—ulcerous destruction of the vessel*. This occasions hæmorrhage, which, according to circumstances, is either external or directed into the tissue—a result which very rarely occurs in spontaneous arteritis, although frequent in the suppuration arising in an artery after it has been tied.

Arteritis is very rarely fatal through the fever by which it is accompanied, or the various inflammations to which it gives rise in important organs. Inflammation of the umbilical arteries, when it extends to the peritoneum, often proves fatal through peritonitis. Spontaneous gangrene in the form of mummification, commonly known as *gangrena senilis*, is often produced during arteritis, or occurs as one of its sequelæ, when it attacks the trunk of a vessel belonging to a part of the body, which cannot be supplied by any collateral circulation. This result of arteritis, which is dependent on the closure and obliteration of the vessel, has been most frequently observed in the lower extremities in inflammation of the femoral arteries. It is a common cause of the fatal result of spontaneous arteritis.

General infection of the mass of the blood, as the consequence of the absorption of the products of arteritis into the blood, and of the secondary metastatic processes in the capillary system with which it is intimately connected, is, according to our experience, a *very unusual phenomenon*, and hence a very uncommon cause of the fatal termination of arteritis. Our own opinion is confirmed in reference to this point, by the concurrent testimony of other observers. (Hasse.)

We have already endeavoured to explain this rarity of the secondary processes, as compared with their frequency in phlebitis. We will here briefly observe, that this rarity must be referred to the greater susceptibility of the arterial blood for taking up inflammatory products which speedily give rise to coagulation and to obturation of the vessel, and to the circumstance that their reaction in the arterial current, being exhausted towards the capillaries in ordinary cases, hinders the general infection of the blood beyond the limits of those vessels. The inflammation of the veins accompanying an inflamed artery, which we have had very frequent opportunities of observing, does not appear to us always to possess the character of secondary phlebitis, occasioned by coagulation of the blood

from its absorption of the products of arteritis, but rather to depend on the transmission of the inflammation from one vessel to another.

This arteritis is of very rare occurrence when compared with phlebitis.

It is occasionally an idiopathic affection, but more frequently it appears to be secondary (metastatic), as it occurs after different acute diseases. The causes on which it depends are frequently very obscure, but in some cases it may be referred to traumatic influences. The form of arteritis which is occasioned by operative agencies, such as ligature, torsion, &c., will, like the process of healing by which it is followed, constitute the subject of future remarks.

General arteritis, like general phlebitis, has no existence.

The above remarks apply to the inflammation of the arteries of the aortic system. In the system of the pulmonary artery appearances indicating inflammation of the larger branches are very rare, and probably are merely secondary processes arising from spontaneous coagulation of blood, and resembling secondary phlebitis. The occlusion of these vessels in most cases results in death before the development of an excessive inflammation in their coats.

The smaller the arterial vessels are, the more dependent are they on the condition of the surrounding tissue. They likewise participate in this inflammatory process, which either penetrates directly into the vessels of their cellular sheath, or affects them indirectly in consequence of the inflammatory products penetrating through and saturating the delicate, permeable coat of the vessel. Hence arise the occlusion of the arteries of an inflamed parenchyma, and the obliterations arising from inflammation terminating in induration, as we see in the walls of healing cavities of the lungs.

Although there exists no true *chronic arteritis* of the form here indicated, acute arteritis having products of an adhesive nature may persist for a prolonged period; the textural metamorphosis of its products may be effected slowly, and the terminations we have already indicated may be only very gradually brought about; while arteritis with purulent exudation may terminate in protracted ulceration. That which is commonly regarded as chronic arteritis or as one of its sequelæ,

is not originally or essentially inflammation, although it constitutes one of the most frequent and most important diseases of the arteries, as we shall presently have occasion to show.

There is, however, a *chronic inflammation of the arteries, manifested as inflammation of the cellular sheath of the arteries (which consists of a layer of elastic, and a layer of cellular tissue), to which its products are limited, and which merely exerts a secondary disturbing action on the normal relation of the inner arterial coats of arteries, viz., the circular fibrous, and the true lining membrane.* This constitutes a very frequent, and, at the same time, a highly important phenomenon in arteries of large calibre, as, for instance, the trunk of the aorta and its branches.

It is occasionally a *primary*, but more frequently a *secondary* disease, and as such constantly accompanies the morbid deposition on the inner coat of the vessels and its metamorphoses. Its anatomical appearances are in general those of chronic inflammation of the cellular tissue, as, for instance, unusual vascularity of the cellular sheath, with dilatation of the injected vessels, and, corresponding to the degree of its injection, a more or less uniform, saturated coloration, varying from a dark red to a purple, while the cellular sheath is infiltrated with a greyish, or greyish red, watery or adhesive and gelatinous fluid.

This disease *terminates* in hypertrophy, thickening, and condensation of the cellular sheath, which is converted into a tough, apparently lardaceo-fibrous, and callous white stratum, varying in thickness from 3 to 6 lines. (Sclerosis.)

It *induces* paralysis of the diseased tissue, more especially of the elastic layer of the vessel, and consequently dilatation, which appears, according to its degree, either diffused, local, or partial. The circular fibrous coat, which is loosened in texture by the dilatation of the vessel, exhibits a morbid brittleness in cases of callous condensation of the cellular sheath, is stratified in appearance, and is of a dirty yellowish, faded colour, which indicates a tendency to spontaneous lacerations, owing very probably to the deranged nutrition of the coat of the vessel. Dilatation of the vessel moreover induces the excessive formation of an anomalous inner coat, and its further consequences.

This inflammatory process and the modes of its termination that we have already indicated, are accompanied by two different conditions of the cellular sheath.

In *the one case*, the vascularised, infiltrated, cellular coat of the artery, together with the elastic layer, admits of being detached with unusual readiness from the circular fibrous coat—a condition which may degenerate into spontaneous detachment, and give rise to spontaneous laceration of the lining membrane (dissecting aneurism).

In *the other* and more commonly observed case, the callous and thickened cellular sheath has coalesced with the circular fibrous coat. This most commonly occurs in dilated aneurismal arteries.

Special reference must be made to *the inflammation of the cellular sheath at the origin of the two arterial trunks, which depends on pericarditis*. It would appear from the milk-spots and adhesions found at this point, that inflammation of this portion of the pericardium occurs very frequently, either partially or associated with general pericarditis. By affecting the subserous cellular substance, it also implicates the cellular coat of these arterial trunks. This inflammatory condition very frequently extends beyond the period of the acute pericarditis, and in the form of chronic inflammation of the cellular sheath gives rise to dilatation, more particularly of the aorta.

In reference to the pulmonary artery, this chronic inflammation sometimes attacks the trunk and its two branches, and it is commonly present in an inferior degree associated with a similar condition of the cellular sheath of the aorta.

b. Ulcerous Processes or destructive ulcerations occur under different conditions in the arteries. They are somewhat frequent, considering the very striking integrity exhibited by the arteries in the midst of extensive abscesses. They invariably originate in the cellular sheath or its vicinity, and in no case do we meet with an ulcerous process either in or upon the inner coat of the vessel. The so-called atheromatous process, which is frequently regarded as ulceration of the inner coat of the artery, is not of an ulcerous character.

To this class belong:

1. The already described suppuration of an artery (see page 257) resulting from arteritis producing purulent exudation—the suppuration arising from arteritis occurring after the application of a ligature, and which will be considered in future page.

2. Small arterial vessels, together with the capillaries, frequently suppurate when there is suppuration of the different tissues, and we then generally find them in a state of obturation, (See p. 258.) In less frequent cases, a very violent suppuration of a low character affects vessels in which there is no occlusion, and occasions hæmorrhage into the abscess.

3. Ulcerous destruction of the larger arteries in the form of circumscribed corrosion and perforation of the arterial wall from surrounding and contiguous abscesses, constitutes a very important and remarkable phenomenon. The wall of the artery is so much destroyed at a circumscribed spot, that it generally presents a round or oval opening, surrounded either by a smooth, as if cut, edge, or a jagged and fringed margin, and contracted in some instances towards the interior in a funnel-like shape; it is attached by means of this aperture to the abscess, or to the base of the ulcer. This form of ulcerous destruction is the origin of many very dangerous external and internal hæmorrhages. Among the most important of these we may instance ulcerous openings of the larger arteries in many different parts of the body. We have ourselves frequently observed perforation of the femoral artery from a suppurating syphilitic bubo, and Hasse noticed such a perforation of the vertebral artery of the right side from an ichorous abscess arising from syphilitic caries of the cervical vertebræ; and to these examples we may add the opening of different arteries on the base of perforating ulcers of the stomach, and the opening of branches of the pulmonary artery by tuberculous caverns.

These ulcerous processes, as we may sometimes notice in the ramification of the pulmonary artery in the walls of tuberculous cavities of the lungs, also give rise to a laceration of the artery. This is owing to the removal of the surrounding protecting parenchyma, and to a loosening and softening of the coats of the vessel, resulting from imbibition of the ulcerous fluid. In some cases this is preceded by a lateral dilatation of the artery towards the cavern.

c. Excessive Deposition of the Lining Membrane of the Vessels.
—We rank with the above anomalies a process which, although it does not originally exhibit a diseased textural condition of the arterial coats, at all events results in such, and moreover

stands in a most relation to chronic inflammation of the cellular sheath of the artery, the latter being either associated with it, or in rare instances the indirect cause of its origin, in consequence of previous dilatation of the vessel. It further constitutes the basis of aneurismal formations and of numerous spontaneous obliterations. It is the most frequent form of disease affecting the arteries, and is on that account of the greatest importance. It consists in an excessive formation and deposition of the lining membrane of the artery derived from the mass of the blood, and at the same time constitutes hypertrophy of this membrane.—We purpose devoting the following remarks to the consideration of this subject, in which we will endeavour briefly to notice all its most important bearings.

In a highly developed form of this affection, we find the inner surface of a large artery, as the aorta, covered with a foreign substance spread over it at separate points, or in large patches, and forming a stratum varying in thickness, by which the inner surface of the vessel is commonly rendered uneven. This substance is in some places either greyish, greyish white, faded, and translucent, or in others milky white, opaque, and similar to coagulated albumen; in some rare instances it is coloured by the imbibition of hæmatin over various extents of surface. Its free surface is at the same time smooth and shining, or dull and as it were wrinkled. It is soft, moist, and succulent in the translucent parts, and dense, dry, tough, and elastic in the more opaque portions, resembling a cartilage or fibro-cartilage, with which it is usually compared, and for which it is still occasionally mistaken. In the latter condition it adheres internally to the circular fibrous coat.

This substance admits of being split into lamellæ, and drawn away in the form of strata. If this is done at those spots where the deposition forms isolated plates or islands, we discover that one or more of the lamellæ thus drawn away, generally the innermost (superficial) ones, terminate beyond the limits of the plates in a delicate membrane, which is prolonged to the contiguous, and apparently normal lining membrane of the vessel.

The thickness and extent of this deposition correspond to the degree of the anomalous condition. It varies in thickness from a quarter of a line to two lines and upwards; and extends

in extreme cases over the whole trunk and main branches of the aorta, implicating the entire arterial system.

The deposition is generally the thickest directly over the division of a trunk, or at the bifurcation of a vessel. At these points the deposit is frequently so thick, that the mouths of the divergent vessels are much contracted, and even wholly closed. —The spot at the trunk of the aorta, which next to those we have already mentioned, deserves a special reference as a common locality for this deposition, is the lower wall of the aortic arch adjoining the left bronchus. This deposition, which is in itself highly interesting, is rendered still more so when it undergoes ossification. For as we shall have occasion to show, the osseous concretion gives rise here to an angular curvature, and a consequent contraction of the tube of the aorta.

We find, on a close examination of the deposit, that it has nothing in common with cartilage or fibro-cartilage, with which it is ordinarily compared and even confounded (*cartilaginescence of the arteries*), and that it actually consists of structures analogous to the layers which constitute the lining membrane of the vessel (the epithelium, fenestrated membrane, and longitudinal fibrous coat).

The circular fibrous coat is found, when compared with the other arterial coats, to be soft, brittle, cleft, and of a faded, dirty brownish colour. The cellular sheath exhibits considerable vascularity and puffiness, or is in a state of sclerosis.

It will be seen from this description of the appearances observed in the more highly developed stages of the disease, that our attention should, on the one hand, be directed to its incipient stages, and on the other to its further progress.

At its commencement, this deposition cannot be detected without a previous familiarity with its appearance. It is then a delicate, soft, succulent membrane, exhibiting a vitreous transparency, and appears in some cases, where it is thrown into small folds by the preponderating contraction of the circular fibrous coat, to be exceedingly thin, and covered with white dots or stripes. The circular fibrous coat is normal, when not altered in consequence of pre-existing inflammation of the cellular sheath.

The deposition continuously increases in thickness by the addition of new strata, and thus gradually passes from the

condition of transparency and succulence, characteristic of recent formations, to that state in which it appears opaque, resembling coagulated albumen, and finally presents a ligamentous appearance, having a dull, wrinkled surface.

Before we enter upon the consideration of the metamorphoses which further occur in this deposit, it will be necessary to direct attention to some points which, although of extraordinary interest, have hitherto been wholly overlooked.

On attentively examining the inner surface of a highly diseased artery, we perceive that the *deposit is interspersed with openings or foramina, varying in size from a pin's head to that of a poppy-seed*. These openings occasionally attract attention by a small drop of blood oozing from them on pressure. In some cases these openings are very numerous, whilst in others it is difficult to detect them. They might, at first sight, be mistaken for the contracted mouths of vessels; but the error of this view is readily made apparent by a closer examination, and by the circumstance that they occur at spots where no such vessels are given off, as, for instance, on the ascending arch of the aorta.

These openings lead to canals, which penetrate to various depths in the deposit, where they either terminate without changing their form, or again divide, and turning, with their branches, in an oblique direction, enter the circular fibrous coat, where they finally ramify. They constitute a system of canals to convey the blood into the deposit and the cleft circular fibrous membrane, which is filled by the blood of the diseased vessel, and may frequently be seen through the deposit.

As far as we are aware, these canals are not connected with the vascular system of the cellular coat, and do not anastomose with its vessels, although they penetrate as far as its elastic layer.

The manner in which these openings and canals originate is a question of the greatest interest. They are undoubtedly the result of partial resorption in the deposit, by means of which openings are formed, which enlarge into canals by coming in contact with each other in the different strata of the deposit. Their mode of origin is very probably similar to that of the apertures of the fenestrated or striated arterial coat, and is

closely connected with it; it is very likely, also, the same process which Stilling observed in the thrombus of tied arteries, and which we have also noticed in a fibrinous coagulum in the heart (in the so-called polypus of the heart), where it rendered the coagula porous, and caused them to acquire a cavernous structure, erroneously regarded as a condition of vascularity.

This *channeling* of the deposit undoubtedly constitutes the basis of that degeneration of the arterial walls which Lobstein considers under the head of *softening of the arteries* (artério-malacie). The wall of the artery, in some few cases, degenerates into a spongy tissue, resembling the corpus cavernosum, or occasionally into the form of a tumour, from which, when it is cut, blood pours forth from an innumerable quantity of openings, as from a sponge.

The metamorphoses through which the above described deposit passes, after it has become completely opaque, are the so-called *atheromatous process*, and *ossification* of the arteries.

1. *The atheromatous process* consists in the metamorphosis (disintegration) of the deposit into a pulpy mass, compared by the French to a purée of peas, consisting of a large number of crystals of cholesterin, fatty globules, and of molecules exhibiting various degrees of consistence, from coarseness to extreme fineness, and consisting of albumen and calcareous salts.

The metamorphosis begins with a finely punctated opacity and decoloration of the deposit, and is not limited to any definite duration, occurring sometimes at an early stage, and at other times at a more advanced period, although, as has been already observed, generally when the deposit has become opaque. It, moreover, commonly begins in the deeper, older strata of the deposit, and advances from thence towards the surface. It usually affects a space varying in circumference from the size of a lentil to that of a crown or a shilling piece. There is, at the same time, an increase of volume, and a swelling of the deposit; the uninjured lamellæ rising above the surface towards the interior in proportion to the depth to which the process has affected the deposit, and then frequently exhibiting a perceptible fluctuation.

After this process has penetrated to the innermost layers, or when they have burst above the pulpy mass, and been torn

asunder by the force of the blood pressing into the cavity, the mass itself appears uncovered on the inner surface of the artery, and in contact with the blood, in which case fibrinous vegetations of different forms are deposited on the fringed margins, after the occurrence of the bursting or rent.

The pulpy mass, both immediately after it has been laid bare and also subsequently, is taken up in different quantities into the blood, although another and the more consistent part of it is infiltrated by the blood, and permeated by its fibrin, and is thus rendered firmer, and, at the same time, coloured by hæmatin, in various degrees of intensity, being first of a dark red, then of a dirty brown or purplish, and lastly of a yeast-like colour. In addition to these discolorations, the mass acquires a very peculiar appearance, when its surface is covered with large crystals and accumulations of cholesterin, for it then looks as if it were interspersed with spangles, or silver-like and shining scales.

These spots are even at the present day regarded as *ulcers—fungal ulcers of the arteries*. But the atheromatous process presents no essential analogy with an ulcerous process, nor is the deposit itself an inflammatory product. We discover no trace of an ulcerous product in the atheromatous mass, and its admixture with the blood is not characterised by any marked subsequent symptoms, as we learn from the experience of hundreds of cases.

The atheromatous mass is very often gradually thickened, and converted into a moist, soft, plaster-like substance; and finally appears in the form of a coarsely granular stalactitic calcareous concretion.

This loss of substance is occasionally replaced by a fresh deposit, when the atheromatous mass has either been wholly, or for the most part, taken up into the blood, in which case these spots remain below the level of the inner surface of the vessel, and thus acquire a cicatrix-like and wrinkled appearance, in consequence of the amount of shrivelling of the callous cellular sheath. They also very often acquire a slate grey or greenish grey, or black colour from the hæmatin by which the tissue is saturated, and which remains on the margins and on the base. They are regarded as *cicatrices of the supposed ulcers of the arteries*.

2. *Ossification is the second form of metamorphosis of the deposit.* This includes the well known ossification of the arteries. It presents many essential points in common with the atheromatous process, and occurs only in a deposit of comparatively extensive thickness, beginning in the lowest strata, where it is first manifested by the pale turbidity of the deposit, which has then become opaque.

When the process of ossification has been completed, and the metamorphosis has extended throughout the whole thickness of the deposit, the osseous concretion lies exposed. Its form is that of a concavo-convex plate, having a tolerably smooth, even, and concave inner surface, and a rough, nodularly uneven, convex external surface, with irregular and jagged margins. The bony plate is bored through at the point where a branch of the ossified artery is given off, unless the mouth has already been previously closed by the deposit. In arteries of large calibre, as the femoral arteries, the concretions present the form of rings, which enclose more or less of the circumference of the vessel.

The *number* and *size* of these concretions are subject to great variations; in some cases they only appear at detached points, whilst in others the artery appears to be converted into a more or less solid osseous tube.

In large arteries, the exposed bony plates are often partially detached by the current of the blood, when they remain in the vessel at different angles of inclination. Their rough margins readily become the seat of fibrinous vegetations.

This form of arterial ossification exhibits a yellow colour, and, in general, considerable density and hardness of texture. It is deficient in the delicately lamellated structure of bone, and has no medullary canals or bone-corpuscles. (See Miescher, Valentin.)

The seat of arterial ossification is the lining membrane of the vessel, which is itself produced in anomalous excess. The bony plate does not, according to the general view, remain stationary between the inner and the circular fibrous coat; nor does it press these layers asunder, and induce atrophy in the former of the two by pressure, but it is developed in the parenchyma and in the numerous thick superimposed strata composing the recently formed inner coat of the vessel. It at length becomes

exposed in the artery, in consequence of the final ossification of the innermost lamellæ, and not simply from their atrophy.

Besides these, other concretions are occasionally observed in the arteries. Thus, for instance, granular or stalactitic calcareous masses are occasionally found upon the inner surface, or the raised margin of these bony plates. These are either thickened, cretified, atheromatous substances (See p. 266), or cretified fibrinous vegetations.

According to our view, although in opposition to that of many good Pathologists, these two processes or metamorphoses very commonly co-exist; but, it must be admitted, without any marked preponderance in either of them.

Having given the above description of this deposit and its metamorphoses, and having observed that all the different conditions of this deposition very frequently exist in the same vessel, we purpose, in the following remarks, to consider *the relation simultaneously exhibited by the other coats of the arteries.*

1. At the commencement of the deposition, and till it attains some degree of thickness, there is no perceptible alteration in the *circular fibrous coat*. But such alterations become the more apparent in proportion to the increasing thickness of the deposit; for this coat gradually acquires a dirty yellowish colour, its texture becomes looser, and it admits, with uncommon readiness, of being separated into fibres and layers; at the same time it loses its elasticity, and yields to the pressure of the column of the blood, whence dilatation of the vessel supervenes to a degree that corresponds to the extent of inflammation already set up in its cellular sheath. It loses its power of resistance, and becomes thinner in consequence of this dilatation.

In the more highly developed stages of this deposit, and when the atheromatous and ossifying processes have become fully established, and even made considerable progress, the circular fibrous coat presents a dirty brown, yeast-like colour, and is soft, lacerable, and cleft. As the dilatation of the vessel increases, the fibres gradually separate, and the deposit sinks into the interstices thus produced, where it comes in contact and finally coalesces with the cellular sheath, which has in the mean time been converted into a callous tissue.

According to our observations, this disease of the circular

fibrous coat depends on the development of fat—fatty degeneration, by which, analogously with the process observed in the so called fatty metamorphosis of the muscular tissue, its peculiar ramifying fibres and its elasticity are destroyed.

This coat is directly implicated in the atheromatous process, which extends to it from the deposit, and destroys it.

2. *The cellular sheath of the vessel, in the majority of cases, is found to be in a state of chronic inflammation*—that is to say, in a state of vascularity, redness, infiltration, and puffiness, or has been converted, in consequence of this process, into a layer of white, very dense, callous tissue of considerable thickness, coalescing with the circular fibrous coat, or with the deposit within its interstices. The following points are of the greatest importance in reference to this condition of the cellular sheath :

a. The intensity of this condition bears no relation whatever to the degree of the deposition, since, in the higher stages of this process, it is occasionally, and in the less developed stages very frequently absent, whilst it never exists in the incipient form of the deposit.

b. This condition must, therefore, be of a *secondary* character, and associated with a certain stage of the deposit. This fact does not, however, exclude the possibility of the converse relation, for as we shall have occasion to show, a primary and substantive chronic inflammation of the arterial sheath may give rise to a local deposit, in consequence of dilatation of the vessel.

The deposit is either *local*, limited to one or more spots of the vessel, or it extends over a large portion of an artery, or over a separate part or the whole of the arterial system. In the former case it depends on local dilatations of the vessel, and on the slowness or partial stagnation of the current of the blood; in the latter, the controlling influence must be a general state of disease, which we would designate as a *constitutional* condition.

The deposit, when appearing as a constitutional disease, occurs almost exclusively in the *arteries*, and only in the *aortic system*. This agrees with the consecutive anomalies, especially the occurrence of ossification of the vessels, aneurismal formation, and obliteration.

Very little relative importance can be attached to any scales purporting to give the frequency of the occurrence of this constitutional affection in the different portions of the aortic system, for whenever the disease appears especially developed in any definite part, the rest of the system—as, for instance, the aortic trunk—will also be implicated.

The trunk of the aorta is most frequently the seat of the disease; and here we find that the ascending aorta, and the arch, are most commonly affected, next the abdominal, and lastly the thoracic portion.

Next in order follow the splenic, the femoral, the internal iliac, the coronary arteries of the heart, the trunks of the arteries of the brain—that is to say, the carotids within the cranium and the vertebral arteries, with their branches,—the uterine, the brachial and subclavian, the spermatic, the common carotid, and the hypogastric arteries.

It is worthy of notice, that certain arteries are only very rarely, and in exceptional cases, subject to even a subordinate degree of this disease; among these we may reckon the mesenteric arteries, and yet more, the cœliac, the gastric, the hepatic, and the epiploic.

This scale corresponds generally to the frequency of the occurrence of metamorphoses in the deposit, such as its ossification, as well as aneurismal formations.

We have not been able to determine, with the requisite accuracy, whether there actually exists such a symmetrical occurrence of the conditions already described (viz., crude deposition, ossification, and the atheromatous process) in the corresponding arteries of the two sides of the body, as Bizot maintains that he has observed, and regarding which he has established a law; and indeed our views of the constitutional character of the disease prevent our attaching any great importance to the subject.

This disease is of very rare occurrence in the *pulmonary artery and its branches*; but if it be present here, it is always likewise considerably developed in the aortic system.

This affection scarcely ever occurs as a constitutional one in the *veins*, for here it always exists as a secondary phenomenon, depending on a sluggishness of the current of the blood, produced by ordinary causes. (See Diseases of the Veins.) On the

other hand, the veins are frequently affected by a morbid formation, which, although it may not be purely constitutional, yet presents a very remarkable analogy with the disease of the arteries under consideration: we refer to the so-called *phlebolites* or *vein-stones*. It is, moreover, worthy of special notice, that the deposit in the veins commonly attains a high degree of development when arterial blood makes its way into them. (Varicose Aneurism.)

Sex exercises no special influence on the occurrence of the deposit and its different consecutive conditions; but Hasse appears, on the whole, to be correct in his view, when he states that disease of the abdominal aorta is more frequent, and more highly developed, in women than in men.

Age, on the other hand, gives rise to important differences; but although the disease is most frequent between the fortieth and the sixtieth year, the assertion that it increases in frequency in proportion to the age, and that it occurs in advanced life almost as a normal condition, is not well grounded; for although it may undoubtedly date in many aged persons from a comparatively early period of life, there are many others in whom it is entirely absent. Old age presents, indeed, a mechanical disposition to this affection, from the dilatation of the arteries common to that period. Before the above mentioned age, the disease is undoubtedly more rare, although frequent even between the thirtieth and fortieth year. Before that period it is very much rarer; and when it occurs prior to the age of twenty years, it is mostly only a local disease, depending on congenital or early acquired anomalies of the trunks of the vessels and of the heart. This observation refers especially to its occurrence during the periods of puberty and childhood.

If, after considering the above remarks, we proceed to the question—In what consists the nature of the disease? we gather the following facts from our examination of all the important points bearing on the subject:

1. *The deposit cannot be regarded as the product (exudation) of an inflammation of the arteries.* The chronic inflammation of the cellular sheath of the diseased vessel is almost always a secondary consecutive appearance which associates itself with the already established deposit.

2. *The deposit is an endogenous product derived from the blood, and for the most part from the fibrin of the arterial blood.*

3. *Its formation demonstrates the pre-existence of a peculiar crasis of the blood, which is intrinsically arterial, although at the present time we are wholly ignorant of the character of the peculiarity on which this depends. We must regard the old dogmatic view, which sought the cause of the affection in arthritis, as an opinion deficient in proof.*

4. *In proportion to the extent of the disease of the arteries, so much the less likely is it to be combined with tuberculosis; and this disease undoubtedly is in part the cause of that immunity against tuberculosis which we constantly notice in large aneurisms of the trunk of the aorta. The grounds of this relation are not known; but it is not wholly improbable that this immunity may arise from a similarity between the process of deposition (which occurs in the form of separation of fibrin,) and the tuberculous process, by exhausting the arterial character and the materials of the blood. On the other hand, we very frequently observe an excessive production of fat associated with the deposition and ossification in the arteries. This abnormal formation occurs—independently of the fatty degeneration of the circular fibrous coat, and of the atheromatous process,—more especially in the neighbourhood of the ossified arteries with atrophy of the muscular tissue, in the vicinity of aneurisms, and, in addition, as excessive accumulations of fat in the blood, of cholesterin in gall-stones, &c.*

5. *The deposit and its metamorphoses present numerous, highly important analogies, that have hitherto been wholly neglected. For the sake of brevity we will here notice only the most important; viz., the deposit also occurring under certain conditions in the veins, the phlebolites, (which we will consider under Diseases of the Veins,) the capsules investing different fibrinous coagula in the vascular system, and causing them to adhere to the walls of the vessels and of the heart, and the metamorphosis which these fibrinous coagula undergo within the vascular system, and which may even affect fibrinous coagula externally to that system.*

The *effects* produced by this disease in its reaction on the whole organism are still unknown. In respect to the vessel itself, the disease gives rise to different forms of dilatation, with

contraction of its branches and complete closure of their mouths, constituting a highly important, although little known, secondary condition. When arteries of lesser calibre have been ossified, and the deposit continues to exist, they finally become closed and obliterated. It is, moreover, probable that the capillary arteries at the seat of the deposit become diseased, in consequence of the diminution and cessation of nutrition arising from the obstruction and arrest of their permeability. Either may, moreover, give origin to the formation of spontaneous gangrene—the so-called dry gangrene—mummification of the tissues. Finally, this disease is often found to terminate in spontaneous laceration of the large arteries, and especially of the small trunks.

It still remains for us to add the following remarks to the observations already made in reference to the diseases of the valves of the heart at p. 227. The valves of the aorta exhibit a thickening and an adhesion to the wall of the vessel, and appear fused together with consecutive shrivelling, malformation, and ossification. We have already remarked, in the same page, that this disease which generally forms the basis of that insufficiency of the aortic valves which is slowly and almost imperceptibly developed in advanced life, is not of endocarditic origin, but depends upon an excessive formation of a tissue analogous to the inner coat of the vessel, and deposited from the blood upon these valves. It is commonly associated with a diffused deposition in the trunk of the aorta and a dilatation of the latter with aneurismal formation.

d. Adventitious Products.—A very few of these forms occur in the vascular system generally, and especially in the arteries.

1. Among the productions of *fibroid tissue* we may include sclerosis of the cellular sheath in consequence of its chronic inflammation, and perhaps also that metamorphosis of the deposit in which it becomes converted, in many cases, into such a tissue.

2. An *anomalous production of bone* occurs in the following forms:

a. The well known ossification of the arteries—a metamorphosis of the deposit—to which we have already sufficiently alluded. The frequency of its occurrence in the different portions of the system, corresponds with that of the morbid de-

position in the arteries on which it depends. A similar relation exists in reference to the periods of life and the sex in which it most frequently occurs.

b. Chalky, mortar-like concretions, formed by the thickening of the atheromatous mass, or by the metamorphoses of the fibrinous vegetations deposited on the above named bony plates, &c.

c. Ossification of the occluding plug, which owes its origin to inflammation with exudation on the inner coat of the vessel, after the previous conversion of the plug into a fibroid string.

3. The numerous forms in which the *anomalous production of fat* occurs are extremely important. The cholesterin which is contained in the so-called atheroma of the arteries, is the only fatty product that has been hitherto noticed. (Gluge, Gulliver.) Yet the fatty degeneration of the circular fibrous coat, (See p. 269,) which may be compared to steatosis of the muscles, is, in our opinion, more important in its results, whilst the excessive formation of fat that is combined with ossification of the arteries, presents numerous points of interest, both on its own account and in consequence of many analogous conditions. (See p. 272.)

4. *Cysts* are of very rare occurrence in the arteries, and probably only appear in the cellular sheath and the neighbouring tissue. We have never yet observed a case of this nature. Corvisart attempted to build up a theory of the formation of aneurism on two cases which he had observed, but his views were long since refuted and never met with support.

5. *Tuberculosis* neither occurs here nor in any part of the vascular system. The deposit does indeed, as we saw at p. 272, give indications of an analogous process, which gives interest to the fact that this disease does not occur in the venous system as a constitutional one.

6. The larger arteries with thick walls steadily resist the invasion of *Cancerous Degeneration*, and in this respect present a striking contrast to the veins. Whilst the veins traversing a cancerous tumour exhibit cancerous degeneration of their walls, and are often completely filled up by cancerous excrescences, the arteries are found to be undestroyed.—While the spontaneous coagulation of the blood in the arteries is rare, we find that the tissue developed from it is still more rarely

of a cancerous nature. Velpeau and others have observed the very rare occurrence of an obstruction of the aorta and iliac arteries by a plug of a cancerous nature, in an individual exhibiting a general cancerous dyscrasia. The rarity of cancer of this form—*primary cancer of the arteries*—is worthy of notice, when contrasted with its more frequent occurrence in the veins.

§ 4. *Anomalies of Calibre.*

A. *Dilatation of the Arteries (Aneurism).*

As we purpose limiting ourselves, in the following pages, to the consideration of the conditions of actual dilatation of the artery, we will postpone to another chapter the consideration of false, varicose, and dissecting aneurisms, as subjects which do not appropriately belong to the present place.

After simply referring to the development of the arteries in reference to their calibre and the thickness of their walls, as observed in organs having become hypertrophied, (in the gravid uterus and in morbid augmentations of size in the most different organs,) we will pass on to dilatations of the arteries based on disease of the arterial coats.

It seems, however, especially necessary to notice, in the first place, the conditions under which dilatation of an artery is established. These are as follow:

1. Some dilatations arise without any visible alteration of texture *in consequence of a loss of elasticity and contractility of the coats of the artery*, that is to say, of the circular fibrous coat and of the elastic sheath, or in consequence of a *mechanically induced continuous and excessive filling of an artery, or even of one entire section of the arterial system*. Such dilatations are very commonly observed, particularly in advanced life, in the trunk of the aorta, and especially in the ascending arch, *in consequence of the first above named causes*. If, as we think not improbable, Cloquet's two cases of *cirroid aneurism* (or *arterial varix*), belong to the class in which the aneurism is not the result of extensive chronic inflammation or of deposition, such a dilatation might be extended over the larger portion, or even the whole of the arterial system. In such dilatations the walls of the arteries are thinner, softer, and

more yielding, and the circular fibrous coat is paler than usual. The dilatation, especially at the trunk of the aorta, excepting where it exhibits a more or less strongly marked protrusion of the convex portion at the ascending aorta, presents a regularly cylindrical form: in the so-called *circoid aneurism*, however it appears so far irregular that it preponderates in one or other portion of the surface, and thus gives rise to the interlaced, twisted course of the artery, (which seems as if it were coiled round an imaginary axis,) and to the bulgings occurring at certain points, as well as to the lengthening of the diseased vessel.

Dilatations arising from mechanical obstructions are observed in the aorta; but more especially in the pulmonary artery and its branches, in consequence of the various diseases of the parenchyma of the lungs which impede the normal injection of the capillaries, in cases of stenosis of the left side of the heart, &c.

2. *Chronic inflammation of the cellular sheath of the arteries* gives rise, as has been already remarked, to dilatation of the vessel, in consequence of its paralysing the elastic coat. This is especially observable in the trunk of the aorta, and it may, moreover, have laid the foundation of some or other of the recorded cases of *circoid aneurism*. Dilatations of the point of origin of the trunks of the pulmonary artery and the aorta, depending upon inflammation of the cellular sheath, combined with pericarditis, deserve notice on account of their locality. (See p. 152). These dilatations are generally of a cylindrical form.

The forms of dilatation based upon the two causes above described, constitute (more particularly in reference to the arterial wall enclosing the dilatation) *true Aneurism* (*Aneurysma verum*), which has been distinguished by the designation *Arteriectasis* from those aneurisms which are regarded as depending for their origin on a more essentially anatomical disturbance.

3. *Most forms of dilatation commonly included under Aneurysm (spontaneous aneurism), and at the same time the most important of all, are owing, as we have already shown, (p. 262,) to the deposition of a tissue, analogous to the lining membrane of the vessel, derived from the blood, and occurring upon the inner surface of the artery, and to consecutive disease of the*

circular fibrous coat and the cellular sheath. Attempts based on an examination of the mode of construction of their walls, have been made to separate these dilatations into *true*, and *false* or *spurious aneurisms*; or in accordance with a principle at variance with Scarpa's view of the cellular sheath of the vessel, into *true* and *mixed aneurism*, the latter being subdivided into *external mixed* and *internal mixed* or *hernial aneurism*. This class, moreover, comprises the dilatations which Cruveilhier and others, distrustful of the results of anatomical examination, divided, merely in accordance with their external forms, into *diffuse*, *fusiform*, *cylindroid*, and *sacciform aneurisms*. The last class were divided by Cruveilhier into "*A. sous l'aspect d'ampoules*" with subdivisions into "*A. périphériques*, *semipériphériques*," and "*à bosselures*," and "*A. sous l'aspect de poches à collet*."

These dilatations considered under the common term of aneurism—spontaneous aneurism—will form the subject of the following remarks.

We must, however, begin by observing that a classification of aneurisms, simply based on the anatomical conditions of the coats of the artery, can only have reference to *gradual disturbances*, and cannot, therefore, afford a representation of essentially different and well defined species, where the grounds that give rise to the formation of aneurism are the same. Although, indeed, a division derived from external form may have some practical utility, it cannot afford a sound classification, inasmuch as it has no reference to the anatomical disturbance on which the form depends, nor can it separate well defined species, owing to the numerous transitions of form which they undergo.—We do not purpose giving any special description of the arrangements made by different observers of these forms, since the references made to them in the appropriate parts of our work will be sufficient for their correct apprehension.

Spontaneous Aneurism appears in its simplest type as a diffused dilatation of a vessel towards all points of its surface in a *cylindrical form*, or when, as is usual, it gradually decreases towards both its extremities, till they assume the normal calibre, it is *fusiform* (the *A. diffusum cylindroideum* of some writers, Cruveilhier's *A. sous l'aspect d'ampoules périphériques*.)

Where the diseased condition of the coats of the vessel affects a more or less sharply defined and considerable portion of the surface, the vessel dilates at that point in the form of an originally shallow pouch, which is gradually converted into a sac, flattened at its edges, where it is in contact with the interior of the artery, constituting *saccular Aneurism*, Cruveilhier's *A. sous l'aspect d'ampoules semipériphériques*. The same is the case when, in diffused disease of the vessel, the morbid condition preponderates at any one point of the surface; the vessel being then generally dilated, but more especially at this point, whereas in the former case it is dilated into the form of a sac with ill-defined margins, and flattened at the spot where it branches off from the vessel.

It will be readily understood that a cylindrical or fusiform aneurism may very commonly be converted into a saccular aneurism, in consequence of the *preponderance* of disease at one or other point of its walls; or, even in the absence of disease, this change of form may arise at any point especially exposed to the force of the blood-wave, as for instance at the convexity of the ascending aorta.

These aneurisms are not only remarkable for their frequency, but more especially for the *extraordinary size* which they very commonly attain. The most important among them are those in which the saccular expansion affecting a portion of the periphery of the vessel is so situated that the wall opposite to the sac retains its normal form and direction. These approach to the pedicled aneurisms.

Saccular Aneurisms are commonly of a round form, although they are occasionally oval or conical in shape, even from their commencement; more frequently the form loses its roundness in consequence of excessive disease of the coats of the vessel at different points, when the dilatation preponderates more or less in one direction.

Finally, *secondary pouches in the form of roundish or conical elevations, or protuberances*—Cruveilhier's *A. sous l'aspect d'ampoules à bosselures*—not uncommonly occur in the aneurisms already described (viz., in cylindroid, fusiform, and sacciform aneurisms), when the disease of the coats of the vessel preponderates at several generally inconsiderable, somewhat sharply defined points. These protuberances

may in their turn give rise to excrescences of a secondary form (*a tertiary aneurismal formation*), which project in various ways over each other, giving the vessel the appearance of an irregularly lobulated sac.

When a cylindrical or fusiform dilatation attacks a vessel irregularly over a considerable portion of its surface, and preponderates at different alternate points of the periphery, causing the vessel to extend in a longitudinal direction, by which its course becomes twisted, and as it were distorted round its axis, we again have the *form of aneurism termed cirroid*.

A study of the development of the above forms yields the following facts in relation to *the construction of the walls of these aneurisms*.

When the dilatation has not exceeded a certain degree, the walls of all these aneurisms consist of the whole of the diseased coats, and hence constitute *true aneurism*. The internal stratum is formed by the deposition in its different conditions of opacity, fibroid metamorphosis, atheromatous process or ossification, and in a state of actual channelling. It represents the lining membrane of the vessel. Next follows the decolorised and cleft circular fibrous coat in the act of being metamorphosed into fat; and, lastly, the elastic and cellular coat—the cellular sheath—whose fibres are entwined with one another, and with those of the circular fibrous coat in a hyperæmic and vascular condition, and which exhibits either a bluish red coloration, or pallor and sclerosis.

When the dilatation is very considerable, the tissue of the circular fibrous coat is found to be much separated, while the stratum seen through it is thinner than usual. When the dilatation has extended beyond a certain limit, the fibres of the circular fibrous coat are not only completely separated, and even wanting at several inconsiderable portions of the aneurism, but this coat gradually disappears at the borders of the highest elevation of the fusiform or saccular aneurism, and at the margins of the superposed secondary pouches; the wall of the aneurism consisting here only of the diseased lining membrane of the vessel and of the cellular sheath. At these pouches the dilatation constitutes the so-called *internal mixed or hernial aneurism*, which we purpose considering more in detail.

There is a form of aneurism presenting considerable interest

in many points of view, which Cruveilhier has designated, *A. sous l'aspect de poches à collet*, and which may be termed *pedicled aneurism*. It differs very decidedly in its marked external form from the saccular variety already described, and is separated from it by many stages of transition.

This aneurism resembles a round sac, which is in general attached to the diseased vessel by means of a neck-like base or contraction. This base corresponds to an opening into the vessel, which is equal in circumference to the contraction, and is either round or oval in form, and surrounded by a projecting margin. It constitutes the channel of communication between the vessel and the aneurism.

No form of aneurism presents such striking variations in magnitude as this; since, without reference to the calibre of the vessel, it may exhibit every possible size, from that of a pea to that of the fist, or even of a man's head. The most common size is from that of a walnut to that of a middling sized apple. These aneurisms are, moreover, distinguished by their tendency to burst when still of very inconsiderable dimensions, as for instance, when they are not larger than a pea or a bean.

An examination of the walls of these aneurisms yields the following facts:

1. *In most cases, the wall of the sac at its base near the opening into the artery, consists of the wall of the artery that has been everted by the aneurism and of all the diseased coats of the artery, whilst the circular margin surrounding the opening is formed by a duplication, as it were, of the entire wall of the vessel.* At different distances from this point, however, the circular fibrous coat, after having become gradually thinner, entirely ceases, and the wall of the aneurism then consists almost wholly of the deposition (the inner coat of the vessel) and the cellular sheath. The margins of the opening are smooth and covered by the deposit; the aperture is roundish.—These appearances present themselves in very small as well as in large aneurisms of this nature. *This form of aneurism almost invariably occurs as a secondary formation, being seated on a cylindroid or fusiform aneurism.*

2. *In some few cases the circular fibrous coat terminates sharply at the margin of the aperture in the artery. The aperture is generally irregular and angularly contracted, whilst*

the wall of the saccular pouch above it consists of the cellular sheath and of a deposit, which projects from the contiguous inner surface of the vessel over the margin of the circular fibrous coat in the form of bridge-like plates and strings, and adheres loosely to the cellular sheath in the cavity of the expanded portion of the vessel. This appearance is observed only in small aneurisms before they exceed the size of a bean or a hazel nut, and they then commonly prove fatal by bursting. They usually occur as primary aneurisms, and in general in arteries that are only slightly and locally diseased. The aperture in the circular fibrous coat is obviously the result of loss of substance.

3. In cases of similarly rare occurrence, we meet with a sharply defined bulging of the artery, filled with the atheromatous mass resulting from the disintegration of the deposit and the circular fibrous coat. *The wall is here composed of the cellular sheath.*

On considering the above relations, we arrive at the following conclusions in reference to the origin and form of these aneurisms.

Although nothing positive can be determined in reference to the question, whether the appearances considered under the first head are the result of the further development of the appearances included under the second head, it is however very probable that such is not the case. The aneurism considered under the first head appears to be the result of excessive disease of the coats of the artery at a circumscribed spot. The artery bulges, and its wall then bends at the margin of the diseased tissue towards the tube of the vessel, with which it forms, as it were, a duplicature of the wall of the artery. Finally, the circular fibrous coat gradually separates at the top of the bulging, when the deposit, consisting of the diseased lining membrane of the vessel, is brought in contact with the cellular sheath with which it coalesces within this cavity. The further enlargement is now especially exhibited at this point, until the wall of the aneurism finally consists, for the most part, merely of the deposit and the cellular sheath.

This is undoubtedly the form that has been named *external mixed aneurism*, (Scarpa's *Aneurysma spurium*), and is believed by some observers to consist merely of the cellular

sheath of the artery, in consequence of their having regarded its investment as unessential and as a recent formation, and from their inability to trace the whole of the layers passing into the aneurism. Since we are disposed from our view of the subject to regard the investment as originally formed by the diseased lining coat of the vessel (although certainly in a condition of expansion and attenuation) which has coalesced with the cellular sheath in the aperture formed in the circular fibrous coat, we may regard this aneurism as the same which has been named by other observers *hernial aneurism*.

The appearances considered under the second head, have undoubtedly been developed from those noticed under the third head; at all events we are unable to discover in what manner this loss of substance has taken place within the wall of the vessel, unless by the atheromatous process described under the third head. We find that the deposit and the circular fibrous coat are affected throughout and destroyed. The atheromatous mass is gradually lost by being absorbed into the blood, and hence the bulging at this point consists of the cellular sheath.—This aneurism, when considered in accordance with the above described mode of origin, is, strictly speaking, an *external mixed aneurism*; but in this form it probably never constitutes the subject of anatomical investigation. Thus, for instance, an inner coat of the vessel is produced in the form of a recent deposit, which renders it difficult, or indeed almost impossible, to recognise and distinguish this aneurism from others, especially when it has existed for a prolonged period. This aneurism does not appear, however, to be of long continuance, but generally bursts while of inconsiderable size. It is, moreover, of very rare occurrence when compared with other aneurisms, and cannot therefore have been the sole means of giving rise to the theory in reference to external mixed aneurism, or of originating the opinion of the frequency of its occurrence.

We take the present opportunity of answering the question, *Whether a rent in both the inner coats of the artery can give rise to the formation of an aneurism of this class?* The belief in this mode of origin has met with almost universal accordance, although, as far as we know, the correctness of the opinion has never been proved by any one. The cavity in the neck or

pedicle of these last named aneurisms has commonly been regarded as a fissure. Yet, as far as we are aware, no such rent has ever been detected, nor have we ever found that a fissure in the inner coats of the artery afforded a basis for the formation of an aneurism. (Compare Laceration of the Arteries.)

We believe that the above observations comprise all the most important points in reference to the form of spontaneous aneurisms and the construction of their walls, however much they may seem at variance with a sharply defined classification of aneurisms in accordance with any fixed principle. By way of completing our observations, we will only remark, that the deposit covering the inner surface of the last described aneurism which is attached by a neck, is also found to be affected by the different conditions of opacity, atheromatous disintegration, and ossification.

There still remain several other appearances in the aneurism which demand our attention.

1. *The cavity of the aneurism very frequently contains fibrinous coagula*, which usually form very distinctly stratified masses. The external and older layers consist of a whitish fibrinous substance, generally more or less deprived of colour, and of a faded appearance. They are dense, compact, tough like leather, and dry. The inner layers constantly become looser, more moist, and coloured, until at length the innermost—those of most recent formation—resemble a recent coagulum of blood. The fibrinous layers frequently exhibit many other conditions of great interest. Thus, for instance, the external, denser layers become, in some cases, converted into a whitish, callous texture, which coalesces with the wall of the aneurism, and very considerably strengthens it. In some cases they present an ossification similar to that which appears in the fibroid exudations, as, for instance, on the serous membranes; while, at other times, they are observed to be softening into a yeast-like yellow or whitish pulp, or a cream-like fluid.

As an important phenomenon which is often presented, we may notice that a recently formed layer of the lining coat of the vessel is inserted at different points between the strata of the fibrinous coagulum, giving the whole mass the appearance of being invested with such a membrane, which is then pro-

longed into the deposit investing the interior of the vessel. We here discover the means employed for restoring and maintaining the continuity of the vessel by closing the cavity of the aneurism with a new layer of the lining membrane of the vessel.

The fibrinous layers in the aneurism fulfil, therefore, no other purpose than that of assisting mechanically to maintain the coagulation of the blood and of its fibrin. They are not the product of an inflammatory process in the wall of the aneurism, nor do they exhibit the character of a malignant growth.

These coagula are not present in every aneurism. As a general rule it may be assumed that, without reference to the size of the aneurism, they will be present in large quantities, in proportion to its distance from the axis of the blood-current, and to the smallness of the communication between the cavity and the calibre in the vessel, when compared with the size of the aneurism. Hence we see the reason why fibrinous coagula are so much more readily and extensively deposited in aneurism of the pedicled form, which presents these two requirements in the most highly developed degree, and why their formation becomes the less easy when the aneurism differs from the above and approximates to the spindle-shaped or cylindrical variety. In these and saccular aneurisms, the formation of the coagula depends only on the extent to which the wall of the pouch recedes from the axis of the blood-current. It is, moreover, natural that fibrinous coagula should occur in larger quantity in large than in small aneurisms of the same form.

These fibrinous coagula derive importance from the obstruction they oppose to the rapid increase of the aneurism and to its early bursting, and in consequence of their causing a wasting of the aneurism, and thus inducing its spontaneous healing.

2. *These aneurisms* differ very considerably in their dimensions, as has been already observed, varying from the size of a pea or bean to that of a man's head, and thus occasionally filling up the greater part of one of the large cavities of the body. In general, the largest aneurisms occur on the large arteries, more especially on the trunk of the aorta; but there is no invariable proportion observed between the size of the aneurism and the calibre of the vessel, for aneurisms fully equal in size to those

which occur on the trunk of the aorta, are occasionally met with in vessels of inferior calibre, as, for instance, the femoral and popliteal arteries.

Large aneurisms experience a very extensive alteration in the construction of their walls, to which sufficient attention has not been paid. Until the aneurism has acquired a certain degree of enlargement, it retains its *primary wall*, whose composition we have already described; but when the aneurism exceeds these limits, and the wall is no longer equal to the expansion, its place becomes supplied by adventitious tissues and structures, either over the whole extent, or at more or less sharply defined spots, corresponding to the direction of the increase in volume. These are the structures with which the aneurism is in contact during its increase, and with which it gradually coalesces. This circumstance explains the reason why aneurisms which only increase very slowly in volume, and therefore are only gradually brought in contact with structures able to compensate for the loss of substance of their walls, may attain so great a size, whilst those aneurisms which are rapidly formed and enlarged, and are, therefore, not brought in contact with many of these structures, speedily burst. These adventitious products are accumulations of cellular substance, serous and fibrous membranes, muscular expansions, &c., together with parenchyma, as, for instance, that of the lungs.

We must distinguish between the manner in which aneurisms of great size lose their primary wall, and the loss arising from the result of detritus—the absorption occasioned by pressure where the aneurism is in contact with bone. Thus we find, that where aneurisms, even of very inconsiderable dimensions, are in contact with bone, the aneurismal wall, together with the periosteum, is partly destroyed and the bone exposed.

3. We have already considered all the essential points in reference to the *form of these aneurisms*. It will be evident that the vicinity of resisting structures may, in various ways, modify the form of the aneurism during its growth. Thus aneurisms on the descending aorta occasionally assume a bilobar form posteriorly, in consequence of the resistance offered by the vertebral column, which causes it to separate into two sacs lying on either side.

4. We find great diversity in the *number of aneurisms*

which may be simultaneously present. In some cases several aneurisms are present together, either on different arteries or in close vicinity to each other on the same artery, so that the tube of the vessel exhibits a row of adjoining and even confluent aneurisms. Large aortic aneurisms are usually isolated, which may be explained, at least in part, by the weakening of the mechanical force through the carrying off of a large quantity of blood towards the aneurismal sac.

5. The greatest interest and the most important results arise from the relations exhibited by the branches passing from an artery affected with aneurism; they consist in narrowing or entire closing and displacement of the mouths, and the consequent atrophy of the vessel. These results are produced by various and often intimately connected means.

a. A highly developed degree of deposition (See p. 272), very commonly gives rise to the important conditions of contraction, and, finally, complete closure of the mouths of vessels opening into the artery affected with aneurism. It more especially affects the mouths of small vessels branching off from the diseased trunk, either at a right or an obtuse angle, as, for instance, the mouths of the intercostal arteries, and of other vessels branching off from the diseased thoracic aorta; although it also not unfrequently implicates the mouths of vessels of larger calibre, as, for instance, those of the carotids, the subclavians, &c.

b. Secondly, the mouths of the branches of the vessels are also rendered insufficient and are displaced by means of the fibrinous coagula deposited on the wall of the aneurism. They have commonly been already contracted by the deposition, or have been rendered insufficient by means of the fissure-like opening, which we shall shortly notice. This imperviousness of the mouths is more especially limited to those vessels which branch off from the diseased trunk, either at right or obtuse angles.

In consequence of the displacement and closure of the mouth, the blood which reaches the branch of the vessel through the collateral circulation coagulates, and the vessel is then obliterated from above the plug to the point where the next branch is given off.

c. Thirdly, the branches passing off from a diseased trunk are rendered insufficient by the round form of the mouth being

contracted and altered into a cleft-like opening, which is frequently rendered still more impervious by the projection of a valve-like margin which inclines backwards in the direction of the heart. This is more especially found to occur in the branches of the arch of the aorta, when the latter is the seat of large saccular dilatations.

d. Finally, there is a mode of obliteration that occurs in the vessels branching off from an aneurism, either independently, or complicated with the above described forms. *This mode of obliteration is the result of inflammation with exudation upon the inner surface of the vessel, and of the subsequent coagulation of the blood.* It appears only in vessels having thin walls, and which are therefore liable to this form of inflammation.

The effect of the aneurism on neighbouring parts is to displace and press upon them, in proportion to their inability to offer any resistance to this pressure. By these means the functions of the injured organs are either partially or entirely obstructed. Thus aneurisms of considerable size may variously contract the space of the cavities of the body, and either diminish the apertures of different passages, such as the trachea, the bronchial tubes, the œsophagus, the arteries and veins, &c., or compress them so powerfully as to render them entirely impervious.

Pressure gives rise in different structures to various alterations which are proportional to the degree of pressure and the capacity for resistance presented by the tissue. Moderate pressure generally occasions inflammation in the contiguous structures, which gives rise to condensation and thickening—*increase of bulk.* When the pressure exceeds a certain limit, it results in atrophy. Both of these results are, however, frequently combined, being found simultaneously present in different parts of the tissue; thus, for instance, the parts in the immediate vicinity of the aneurism may be atrophied, whilst the more remote tissue exhibits a new formation of cellular substance and of fibroid tissue. We very frequently observe that bones which have been exposed to the action of an aneurism exhibit atrophy (*detritus*), whilst various osseous formations—*osteophytes*—occur at detached points surrounding the aneurism, and even sclerosis may be present in the contiguous bony layers.

Yielding membranous expansions in part give way to strong pressure, while their fibres admit of being separated; and in part they become gradually atrophied like cellular tissue, serous and fibrous membranes, muscular coats, &c. Large masses of muscle become pale and thin, and even wholly disappear.

Highly vascular and nervous structures, such as the external investment and the mucous membranes, have their texture so much loosened by inflammation, that they readily tear; or where this is not the case, they become gangrenous.

Parenchymatous structures waste away in consequence of the exudation produced by inflammation, and finally become atrophied.

Vessels are obliterated either in consequence of coalescence, induced simply by perfect compression, or in consequence of inflammation, that is to say, by means of adhesion to a coagulum of blood produced by the inflammatory process.

Nerves undergo atrophy through pressure and tension.

Rigid structures become atrophied in proportion to their deficiency of elasticity. Detritus of the bones is therefore very commonly induced by aneurism, whilst cartilage and fibro-cartilage, as for instance the intervertebral cartilages, are longer able to resist this action. This detritus is most frequently observed in the bodies of the vertebræ, in the ribs and the sternum, the clavicle, and also in the scapula in aortic aneurisms, and is often present in so highly developed a degree, that these bones are entirely destroyed, and the osseous wall of the thorax perforated. The vertebral canal has even been seen opened.

The process of resorption induced by the deposition and pressure of an aneurism on the bones, destroys not only the osseous substance itself, but, sooner or later, the aneurismal wall also, which becomes fused as it were with the periosteum and the other fibrous structures that usually invest the bones. The bone is then either very commonly laid bare, or is only covered by a layer of the deposition investing the aneurism, or by the fibrinous coagula in the aneurismal sac. The exposed vertebral column thus very frequently constitutes a portion of the aneurismal wall. In aortic aneurisms which perforate the anterior or lateral wall of the thorax, the roughened and nodular extremities of the ribs, the clavicle, and the sternum, are almost

entirely denuded on their inner surface, and project into the sac of the aneurism.

The effects of the aneurism are diffused beyond its own immediate locality to distant organs, and even over the whole organism. These effects are as varied in their nature as the influences from which they arise; but in general they occur more rapidly, are more violent and are more extensively diffused in proportion to the size of the aneurism, its relation to a main artery, and its vicinity to the heart.

The pressure on the nerves and their tension occasion variously developed symptoms of neuralgia and paralysis.

The pressure of the aneurism gives rise to a varicose condition of the veins below the aneurism, venosity, cyanosis, dropsy, and inflammations, which frequently terminate in gangrene.

Large aneurisms on the trunk of the aorta have a tendency to produce active dilatation of the heart, and this tendency is the more marked in proportion to their vicinity of that organ. They give rise to this disease either in association with insufficiency of the aortic valves, which is, however, generally the case, or independently of this affection. They also induce general venosity, diffused, as it were, from this point, as from a centre.

The pressure on the arteries, and the occlusion resulting from it in the region of the aneurism, may possibly be unattended by injurious results, in consequence of the establishment of a collateral circulation.

The stasis and coagulation of a considerable quantity of blood within a large aneurism, have the effect of withdrawing so large a quantity from the organism, as to occasion symptoms of anæmia, tabes, a watery condition of the blood, general dropsy, and cachexia. The pressure of the aneurism on parenchymatous structures, and the obstruction of their functions, contribute without doubt to the presence of cachexia, and to the development of its special character.

The following must be noticed in reference to *the modes of termination of aneurism*:

Aneurism very commonly terminates fatally.

This fatal termination is very frequently induced by the results already mentioned, amongst which we may specially place diffused inflammations terminating in gangrene, dropsy

of the cavities of the body, hyperæmia and acute œdema, more especially of the lungs, cachexia, and general marasmus.

Spontaneous opening or laceration, rupture and extravasation of blood from the rent constitute a very frequent, always extremely unfavorable, and indeed very often rapidly fatal termination of aneurism. We would direct attention to the following particulars in reference to this subject.

The tendency to spontaneous opening does not bear a direct relation to the size of the aneurism, for we find that small aneurisms burst more frequently than larger ones.

The direction in which the aneurism opens, and in which the blood emerges, varies considerably. Aneurisms in the limbs open into the surrounding cellular tissue, in consequence of which a large quantity of blood is extravasated into the inter-muscular, subcutaneous cellular tissue, below and between the aponeuroses, the muscular sheaths, &c. Aneurisms of the trunk, and of some of the branches of the aorta, as, for instance, the splenic, open into the large cavities of the body, as the peritoneal sac, one or other of the pleural sacs, or the pericardium, occasioning hæmorrhage into the corresponding cavity and the sub-serous cellular substance. Aneurisms of the cerebral arteries open in a similar manner into the sac of the Arachnoid, and into the tissue of the Pia Mater.

Aneurisms very frequently open into canals, as the trachea, the bronchial tubes and their large branches, and the œsophagus, and more rarely into the intestinal canal and the cavities of the urinary passages. They however very commonly open into other blood-vessels, either arteries or veins, and even into the cavities of the heart, more especially the auricles. Such openings very frequently occur in aortic aneurisms into the trunk of the pulmonary artery and its branches, and into the ascending or descending Vena cava.

Aneurisms that are imbedded in parenchymatous structures do not often open; the hæmorrhage here takes place into the parenchyma, and after the latter has been extensively displaced or perforated in the form of a canal, the blood flows freely into the adjacent serous cavity.

We have seen one instance in which an aneurism of the aorta opened into a tuberculous pulmonary cavity having healed, consolidated walls.

Finally, aneurisms may sooner or later penetrate to the general investments, and open externally.

The manner in which aneurisms open is not the same in all.

Aneurisms which project into a serous cavity burst at that part which, having coalesced with the serous membrane, and become extremely thin from a deficiency of the surrounding tissue adapted to strengthen and protect it, offers the slightest degree of resistance in consequence of the excessive attenuation of its walls. The opening generally occurs at one of the most saccular portions of this wall, and is either in the form of a fissure, or more frequently of a roundish aperture having a fringed margin. The latter appearance induced Hasse to believe that the opening was preceded by a pre-existing and self-induced process of softening, but we have never been able to detect its presence in the numerous observations in which we have been engaged.

In those cases in which the aneurism bursts through the walls of the canals on which it is seated, and opens into their cavities, the process is more complicated. Thus aneurisms open into the trachea, the bronchus, and the œsophagus, when the fibro-cartilaginous and muscular elements, together with the adhering wall of the aneurism, are destroyed by detritus, in consequence of the mucous membrane becoming the seat of inflammation, and tearing in that condition with the aneurismal wall. In other cases, as for instance at the œsophagus, a *gangrenous eschar* is developed in the mucous membrane over the encroaching aneurism, and, by extending over the whole of the aneurismal wall, usually gives rise to extensive opening of the aneurism.

The opening of the aneurism into the cavity of the neighbouring blood-vessels is brought about in various ways. In some cases the aneurismal wall coalesces with the cellular sheath of the adjacent artery in such a manner as to deprive the circular fibrous coat of the latter of its proper support. As the aneurism exerts a stronger impulse on the vessel, the cellular sheath becomes completely separated from the artery, and, consequently, the aneurism and its circular fibrous coat at length burst. The rent is in general large, and presents an angular form in this coat of the artery; it is usually complicated with detachment of the cellular sheath over varying

extents of surface from the fissure. (See Dissecting Aneurism.) In other cases, the cellular sheath of an adjacent artery coalesces not only with the aneurismal wall, but also with the circular fibrous membrane of the affected artery, in consequence of a very chronic process of inflammation, and the slow development of the aneurism. The circular fibrous coat directly coalescing with the aneurism is thus rendered thinner, whilst its fibres separate from one another, and at last wholly disappear at different points. At the point which corresponds with the most marked protrusion of the aneurism into the artery, the aneurism bursts together with the layers of the lining membrane of the artery coalescing with its wall. The rent, as in aneurisms that open towards a serous cavity, is small, fissure-like, or resembles a roundish hole.—The opening of aneurisms into a contiguous vein is effected in the same manner. (*Spontaneous varicose aneurism.*)

We occasionally find in aneurisms imbedded in parenchymatous organs and cellular accumulations, that there is an acute inflammatory process, which hinders the development of a protecting and strengthening callus, and by predisposing the tissue to softening and laceration above the pulsating spot, occasions laceration. In other cases the tissues are separated by the pressure, without the concurrence of any such inflammatory process, and thus give rise to the rupture of the aneurism.

When an aneurism opens outwards on the surface of the body the process depends, as in aneurisms that open into the mucous canals, on a high degree of inflammation in the true skin, occasioning a separation or laceration of the tissue, or on a gangrenous eschar implicating the general investments.

Such openings are very often rapidly formed and *single*, although occasionally we observe *several small perforations*, so that there is at first only a gradual and recurring oozing of blood, until the opening acquires a very considerable size.

Finally, the wall of a cylindrical, spindle-formed, or saccular (true) aneurism, frequently exhibits perforations which are owing to a laceration and detachment of the diseased inner coats from the cellular sheath of the vessel. But this is a subject to which we shall revert when we pass to the consideration of the spontaneous lacerations of arteries.

The above forms of aneurismal ruptures in ordinary cases produce death by hæmorrhage, externally, or into one of the large serous cavities, or into the trachea, the alimentary canal, &c. When the aneurism opens into other vessels, such, for instance, as the arteries in the vicinity of the heart, or into any of the cavities of the heart, the result is in general speedy death, in consequence of the obstruction in the circulation. There are, however, exceptions to this rule; and we find that in some instances, small perforations of the latter kind may exist for a prolonged time without causing death, in which case the aperture through which the communication is maintained, acquires a smoothed, healed appearance, from its margin being invested with a recently formed lining membrane. This is more especially the case when the aneurism opens into a vein, and thus constitutes a basis for the formation of a so-called *spontaneous varicose aneurism*. (Thurnam.)

However unfavorable the ordinary termination of an aneurism may be, instances are occasionally observed in which *the disease takes a more favorable turn, and nature brings about a spontaneous cure of the aneurism*. This result is effected in many different ways, which have been especially considered by Hodgson.

1. *The aneurism may compress the artery on which it is seated in such a manner, either above or below, that it gradually becomes impervious, and is then obliterated with the aneurism*. We attempt to imitate this healing process artificially, by passing a ligature either above or below the aneurism. Such a result can only affect saccular aneurisms, and such as are attached by a neck.

2. *The aneurism may be completely filled with fibrinous coagula above which a deposit is formed, which represents the lining coat of the vessel, and stops the communication between the cavity of the aneurism and the tube of the vessel*. Aneurisms attached by means of a neck, and having only a narrow passage of communication, present the most favorable conditions for this mode of termination. We even observe, in some rare cases, that where saccular or spindle-shaped aneurisms have been completely filled with fibrinous coagula, new formations continue to be deposited upon the former, until at length the whole diseased vessel becomes obstructed. Decrease in the general

quantity of the blood and a diminution of the heart's action must be regarded as the most favouring influences.

In both these conditions the aneurism shrivels and contracts over the coagula, either in the form of a fibroid capsule or of a spindle-shaped cylindrical roll, and is then atrophied.

3. *In aneurisms in the extremities the gangrenous process to which they give rise attacks the aneurism itself, and by exciting arteritis, causes the artery to be stopped up by a coagulum.* The aneurismal sac is thrown off and removed, and the artery obliterated at various parts. In the same manner, abscesses and inflammatory foci in the vicinity of an aneurism may occasion arteritis, accompanied by occlusion, and subsequent obliteration of the artery, by which the aneurismal sac is destroyed, and removed by suppuration.

Spontaneous aneurism generally occurs with the same proportional frequency in the two arterial systems, and in the different portions of the aortic system as the disease of the coats of the vessel on which they are based. (See p. 270.) The relative scales of frequency established by different observers are indeed tolerably accurate; but still many of the results which have been given are incorrect; thus, for instance, the assumption of the great frequency of aneurism of the popliteal artery, is undoubtedly so far incorrect that it includes in the same class with spontaneous aneurisms of the lower limbs, which are certainly not of rare occurrence, many others which very probably were of traumatic origin.

In general, aneurisms are incomparably more frequent in the larger than in the smaller arteries, and their occurrence on the trunk of the aorta is characterised by remarkable frequency.

Aneurisms are, on the contrary, very rarely observed in the pulmonary arterial system, where, as far as we know, they are limited to the trunk. Fusiform and saccular dilatations do certainly sometimes occur in the ramifications of the pulmonary artery, within the parenchyma of the lung, as we have observed near tuberculous caverns; but as they originate from entirely different causes, they do not belong to this class. (Compare p. 261.)

Aneurism of the trunk of the pulmonary artery is scarcely ever present, unless there is at the same time aneurism of the aorta, or, at all events, a tendency to that disease.

Although it may be said in reference to the *sex* most frequently affected, that there is a preponderance in men, it is by no means so considerable as is usually supposed. The *age* at which aneurisms are most common is between the 30th and 60th year; they are of much rarer occurrence between the 20th and the 30th year, and must be regarded as extremely unfrequent, and as exceptional cases, when they are present before the age of 20 years. We must, however, exclude from this calculation all the aneurisms of traumatic origin which have hitherto not been separated with sufficient care.

It has long been supposed that aneurisms are *based on a special aneurismal diathesis*, in consequence of the frequency with which they have been observed to appear spontaneously and independently of external influences, and from the fact that several occur simultaneously or in quick succession to each other in the same individual. Thus incidental and individual cases have led to the idea that these predisposing conditions were to be sought in gout, syphilis, or mercurial cachexia; and this opinion was supposed to derive support from the more frequent occurrence of aneurisms in men, as well as from the period of life at which they are most commonly observed. It was conjectured that this diathesis gave origin to the diseased condition of the texture of the coats of the vessel, and to their loss of elasticity and their softening and brittleness.

We have sufficiently considered the anatomical bearings of the disease affecting the coat of the vessel on which the formation of spontaneous aneurism depends, (See p. 262,) and have drawn attention to *an anomaly of the blood-crisis, which may give origin to an aneurism*. (See p. 272.) Nothing positive is known in reference to this blood-crisis; but the concurrence of the above named diseases with aneurism appears to us to be purely accidental, nor do we think that such individual cases afford sufficient scientific grounds for the connection that has been supposed to exist between these diseases and the aneurism.

It is a very important fact *that spontaneous aneurism never exists in combination with tuberculosis*. This immunity is based on the following grounds:

a. The diseased condition of the coats of the vessel on

which aneurisms depends, constitutes a cause of immunity against tuberculosis. (See p. 272.)

b. Large aneurisms of the aorta give rise to consecutive disease of the heart in the form of dilatation, with a readiness proportional to their vicinity to the heart. It is, therefore, in consequence of the venosity and cyanosis occasioned by the latter disease, that aneurisms of the aorta afford a decided immunity against tuberculosis.

Aneurisms, as we have already seen, have nothing in common with cancer.

It still remains for us specially to notice several particular forms of aneurism.

Aneurism of the Aorta.—The aorta is more frequently the seat of aneurism than any other vessel, and the parts most commonly affected by aneurismal formations, are the ascending aorta, and the arch.

The aneurisms most common on the trunk of the aorta are the cylindrical and spindle-shaped aneurism, the saccular form affecting only one side of the vessel, the pedicled, and even the cirroid aneurism, which is occasionally observed along the entire length of the tube of the aorta.

Saccular expansions very frequently occur on the *ascending aorta* at the sinuses, and especially at those two which correspond to the convex wall of this portion of the aorta. These aneurisms very frequently burst at an early stage into the cavity of the pericardium, and occasionally into the right auricle.—Pericarditis in some instances gives rise to the rupture of aneurisms projecting into the cavity of the pericardium.

Aneurisms are incomparably more frequent on the convex than on the concave side of the ascending aorta.

The same is the case in reference to aneurism of the arch of the aorta.

On the *aorta descendens*, aneurisms within the thorax appear most frequently to proceed from the posterior wall and the sides of the vessel, so that they very commonly implicate the vertebral column and the adjacent thoracic wall.

Aneurisms of the abdominal aorta are usually spindle-shaped and saccular, and are most frequently developed from the anterior and lateral portions.

An extensive series of observations has afforded the following particulars in reference to the remarkable peculiarities presented by these aneurisms.

1. Those aneurisms which arise from the convexity of the ascending aorta, and from the anterior and upper wall of the arch of the aorta in general attain a very considerable size, inclining in such a direction that they touch the right half of the sternum, the costal cartilages, and the ribs of the right side from the first to the fifth or sixth, or even extend to the sterno-clavicular articulation and the right clavicle, finally destroying the parts by detritus, and coming to view externally in the corresponding region of the thorax. It is important to remember that such is their course, from which there are very few exceptions, because an aortic aneurism occurring at the sterno-clavicular articulation and at the right clavicle is very commonly mistaken by the bedside for a subclavian aneurism, which is in general erroneously supposed to be of great frequency.

2. Aneurisms, proceeding from the concavity of the ascending aorta, extend in the direction of the pulmonary artery, or are seated in front of it, towards the wall of the left auricle, and open into one or other of these parts.

Those aneurisms which proceed from the concavity and posterior portion of the arch of the aorta, abut upon the trachea and the bronchi, and in general open into them at an early period, and long before they have attained any considerable volume.

3. Aneurisms of the thoracic aorta commonly first implicate the vertebral column at the part corresponding to the above described points of origin, and destroy it to various extents, and in rare cases, to such a degree, that they come in contact with the *dura mater* of the spinal chord, and even burst into the canal. They moreover diffuse themselves over the posterior wall of the left side of the thorax, and occasionally open freely into its cavity, or, in some rare cases, so completely destroy the thoracic wall as to come to view externally on the back. They very often implicate the left bronchus, make their way into the pulmonary parenchyma, and open into it, or into one of the larger bronchial tubes within the lung.—When they occur on the right side of the vessel, they are situated in the mediastinum and on the œsophagus into which they open.

4. In the very rare cases in which aneurisms of the abdominal aorta burst, their contents are usually effused into the cavity of the peritoneal sac.

On Dilatations of the Ductus Botalli.—The dilatations which in rare cases are observed in the Ductus arteriosus, in every period of life, from the earliest infancy, are simple, and not dependent upon any alteration of texture in the coats of the vessel. They are occasioned by a deficient involution of the duct after birth.

If we except that degree of patency of the Ductus arteriosus, in which, in consequence of a uniformly deficient involution (closure), it remains similar in calibre to a branch of the pulmonary artery in new-born infants, and forms a very secondary cylindrical vessel, we find the following different forms of dilatations, which admit of being referred to an unequally deficient closure of its mouths.

1. In *one case*, when the occlusion of both mouths has once commenced, the process goes on more slowly in one—probably the aortic mouth, whose calibre continues permeable after the other mouth has become considerably contracted. Blood now collects here, dilating the vessel, and gradually coagulating within it, forming a spindle-shaped or round, spherical capsule (aneurism), after which this mouth also is finally closed. This anomaly is unquestionably devoid of importance, and does not lead to any secondary consequences, as the coagulum, and the coats of the vessel over it, gradually shrivel together.

2. In *other cases* the Ductus arteriosus is found to present a funnel-like dilatation from the aorta, and the opening into the pulmonary artery is then surrounded by a torn and fringed margin. That this anomaly is not a true patency,—a persistence of the Ductus Botalli in its original form and significance,—is made evident by the above mentioned relation of the duct, and more especially of its mouths; by its violent reopening from the aorta towards the pulmonary artery, as indicated by the character of the mouth; by the occlusion of the Foramen ovale, which is observed in such cases; and the existence of a current opposed to the foetal circulation, and inclined from the aorta towards the pulmonary artery.

This condition is owing to the relation of the duct and its

openings, that is to say, to the violent reopening of the closed ostium of the pulmonary artery from the dilated aortic portion of the duct; and also to the active dilatation present in these cases in the right side of the heart, which is one of the results of the obstacles produced by the entrance of arterial blood into the current of the pulmonary artery.

On Traumatic Aneurisms.—These are aneurisms which patients refer to some traumatic influence, such as a contusion, shock, or some unusual muscular effort, &c., and which the physician, in the absence of all disease in the coats of the vessel, must regard as having such an origin. Aneurisms of this character especially occur in the arteries of the extremities, and, as we have already observed, they are too commonly included without further inquiry under spontaneous aneurism, when they are undoubtedly of traumatic origin. To this class belong a certain number of aneurisms of the femoral, popliteal, and brachial arteries.

We are here led to inquire what disturbance is set up in the wall of the vessel by the traumatic influence, which can give origin to the formation of the aneurism.

This question is very difficult of solution; for on the one hand we rarely or never have an opportunity of examining the artery immediately after the accident, while on the other an examination of an aneurism, when already developed, does not afford absolutely valid grounds for judging of the original disturbance. It is, however, very probable that this disturbance may be based upon *some traumatic influence inducing paralysis of the circular fibrous coat at the affected spot; destroying its contractility, and causing a separation of its fibres; and occasioning a partial laceration of the coat, not affecting either the integrity of the lining membrane or of the cellular sheath of the vessel.* We are led to this opinion from a consideration of the following facts:

1. We cannot believe that traumatic aneurism can be produced by spontaneous laceration of the lining and of the circular fibrous coat of the artery, and therefore be owing to the dilatation of the cellular sheath at the spot where the rent occurs. We have never, for instance, seen an aneurism arise from a separation of the continuity of the lining and circular fibrous coats when it appears either as a spontaneous or a mechanically induced rent; but, on the other hand, there is always, in these

cases, a more or less violent and extended detachment of the cellular sheath, whether the arterial coats be healthy or diseased, constituting a secondary laceration of the sheath, and effusion of blood over the vessel. (See Dissecting Aneurism.)

2. In consequence of the great toughness and power of resistance of the cellular sheath of the artery, a separation of continuity can only be effected in all the coats of the vessel when the shock or contusion has been such as necessarily to produce extensive and repeated laceration of both coats. The consequence of this would at all events be to produce effusion of blood from the vessel over a considerable extent, giving rise to an evident *false diffused aneurism*, which would also be subsequently apparent in the consecutive condition of *false circumscribed aneurism*.

3. The dilatation developed at the affected spot manifests itself originally as a circumscribed and gradually enlarging tumour which is slowly developed, and shows both by its form and construction the probability of the view we have advanced in reference to the disturbance on which traumatic aneurism is based. This dilatation exhibits either the form of a saccular expansion, or of a pedicled aneurism according to the degree of depression of vitality and loosening of continuity produced by external influences in the circular fibrous coat. Its walls principally consist of the lining membrane of the vessel and of the cellular sheath; in the first form we find remains of the circular fibrous coat between the other membranes, while in the second form the fibres of this coat are separated through external agencies; the lining coat of the vessel adhering within the interstices to the cellular sheath, and gradually protruding through it. The attenuation which the lining membrane of the vessel must necessarily undergo, cannot be directly observed, in consequence of the new membrane which has been simultaneously formed in the aneurism. This second form of traumatic aneurism appears therefore to be a *hernial aneurism*, according to the signification we shall attach to it in the following remarks.

On Hernial Aneurism.—The existence of a *hernial aneurism*, or of an *internal mixed aneurism*, has formed the subject of numerous investigations, from the time of Haller to our own day.

We have already become acquainted with aneurisms, which,

as we incidentally observed, must be regarded as hernial aneurisms, in consequence of the anatomical disturbance to which they owe their origin, and in consequence also of the construction of their walls. It now remains for us more closely to define the sense in which we are led from experience to admit the existence of a hernial aneurism.

If it be requisite for the establishment of a hernial aneurism that it should exhibit a dilatation of the lining membrane of the vessel in the form of a hernia through an opening in the middle and outer coat of the artery, we must wholly deny the existence of such a form of aneurism.

1. The direct experiments of J. Hunter and E. Home showed that the removal of the external coat of an artery did not give rise to the protrusion of the lining membrane in the form of an aneurism. Whether the external coat alone, or that and the middle one, were both loosened and detached, the result was simply inflammation and cure without any alteration in the calibre of the injured artery.

However limited may be the application to be extended for various reasons to the results of these experiments, they are still highly interesting, and must excite our surprise from the opposition in which they stand to the result we should have been led to expect. We are not astonished merely at the circumstance that, after the removal of the outer and the middle coat, the lining membrane did not protrude, but still more that, considering its slight power of resistance, it did not at once give way. The circumstance that the middle and lining membranes were not lacerated after the removal of the outer coat, is very probably owing to the elastic sheath having been left on the vessel, and not removed with the outer coat.

2. To the results of these experiments we must add those yielded by observations on human arteries.

Detachment of the sheath of an artery, which consists of an elastic and a cellular layer, is not attended, as we learn from observations of the so-called dissecting aneurism, by a saccular expansion of the exposed yellow and lining membranes, but by its immediate laceration, both in those cases in which it is owing to external influences, and those in which it has resulted spontaneously from a morbid process. When, moreover, this occurrence is met with in cases where the middle and lining

membranes were observed to be healthy, we are the more led to conclude that it would exist where there is disease of these membranes.

In such ulcerous perforations of the arterial wall from without inwards, as we noticed in the femoral arteries, (See p. 261,) notwithstanding the probably gradual and stratified separation of the different layers of the tube of the artery, we perceive no trace of aneurismal formation at the affected spot.

There is, on the other hand, a form of aneurism very frequently met with, which, when considered in the following sense, represents *hernial aneurism*.

a. In spontaneous, spindle-shaped, saccular aneurism, the diseased circular fibrous coat gradually yields at spots which vary in number according to the size of the aneurism. The lining membrane of the vessel (the deposition) coalesces in the interstices thus produced with the cellular sheath, and wherever these portions are excessively dilated, the secondary aneurismal formation deposited on a *cylindroid, fusiform, or sacciform aneurism*, gives rise to aneurisms of a secondary form, commonly known as *hernial aneurism*.

b. Aneurisms that are attached by a neck, and that are composed for the most part merely of lining membrane and cellular sheath, essentially constitute *hernial aneurism*, in as far as they are produced in the same manner as the above named secondary dilatations.

c. Finally, *traumatic aneurism*, in accordance with the process from which it arises, and which has already been described, is a *hernial aneurism*.

Dubois, Dupuytren, Breschet, and others have undoubtedly taken a similar view of the question, when they maintained the existence of a hernial aneurism. We would, moreover, specially remark, although the circumstance seems sufficiently evident from the foregoing observations, that the inner coat of our hernial aneurism is by no means composed of the original lining membrane of the vessel, but consists almost entirely of newly deposited strata.

We do not, for obvious reasons, regard the establishment of hernial aneurism as a separate class to be essential, nor do we think it possible, in all cases, to separate it strictly from spontaneous aneurism.

B. *Abnormal Narrowness—Contraction—Obliteration of the Arteries.*

The arterial system presents numerous varieties of irregular narrowness, and, moreover, exhibits many differences in respect to its extent and degree. To this class belong *congenital anomalies*.

1. *A Congenital Abnormal Narrowness of the Aortic System*, which is strikingly apparent in the large arteries, and more especially in the trunk of the aorta. This is found in some cases, in adults, to be contracted, particularly in its descending arch, to the calibre of an iliac or even of a carotid artery. This anomalous condition, which is very generally associated with deficient development of the system, and with a striking thinness and softness of the arterial walls, is often overlooked in childhood, and very commonly does not exhibit any distinct symptoms until the period of puberty, when it manifests itself by insufficiency in the calibre of the artery compared to the quantity of the blood, and by dilatation of the heart, more especially of the left ventricle. It most frequently occurs in females, and is combined with retarded development generally, and more especially with smallness of the sexual organs.

Anomalies of various extent, amounting even to entire occlusion, are occasionally exhibited in the trunk and branches of the pulmonary artery, occurring as congenital conditions, combined with, and depending on obstructions in the interior of the heart.

2. *A Congenital deficient Development of separate Portions of the Arterial System*, more especially in relation to the calibre and elaboration of the coats of the vessels supplying undeveloped, stunted parts and organs of the body.

Acquired Abnormal Narrowness appears under many forms, and frequently attains so great a degree as to present complete occlusion of the artery. All the various contractions, and the atrophies in which they result, may be principally referred to a *simple involution of the artery*, to *contraction and obliteration in consequence of disease of the coats of the vessel*, to *occlusion of the artery*, and to *contraction and obliteration depending on pressure on the artery*.

1. *Contractions and Obliterations in the Form of simple Involution of the Artery.*

To this class belong the following :

a. *The contraction and subsequent atrophy which affect the arteries* of organs that are becoming atrophied through accidental or intentional (operative) injuries inflicted on portions of the body which have been previously arrested in their growth.

b. *An obliteration which is very similar to the atrophy of the faetal passages, as, for instance, that of the Ductus arteriosus.* Such an obliteration of an artery is occasioned by the establishment of a collateral circulation, which is especially induced by a congenital narrowness (obstruction) of the artery in question. The vessel becomes narrower in proportion to the progressive development of this collateral circulation, and is entirely closed when the latter is completed. To this class undoubtedly belong many cases of obliteration of the different arteries, the causes of which have not been sufficiently explained, and most certainly *those cases of obliteration of the aorta at its arch beyond the part where the Ramus brachiocephalicus is given off, corresponding to the depression of the Ductus Botalli.*

These cases, which embrace the majority of the observations made on obliteration of the aorta, have hitherto been unexplained, both in reference to the malformations on which they depend and the process giving rise to final obliteration. They have been repeatedly collected and arranged. (Barth, Craigie.)

From the interest which attaches to these obliterations of the aorta we are induced to add the following remarks, which are derived from the observations above referred to, as well as from my own experience.

1. An inconsiderable portion of the arch of the aorta, generally at the part already referred to, becomes sooner or later obliterated. The aorta, that is to say its descending portion, is generally abnormally narrow before the establishment of complete obliteration.

2. The aorta before this point, and the branches given off from its arch exhibit considerable dilatation, which extends from these branches over all their ramifications and anastomoses.

3. The heart is in a state of general dilatation, although the

left ventricle is the special and original seat of the affection, which also extends to the trunk of the pulmonary artery and its branches.

A careful consideration of all the circumstances leaves little doubt that the following theory is correct.

1. *This anomaly is based upon a deficient formation*, consisting in the permanence of the aorta in that early foetal condition in which it constitutes a trunk which merely supplies branches to the head and upper extremities, whilst the pulmonary artery bends round towards the descending aorta in the form of the future Ductus arteriosus, and supplies branches to the rest of the body. The ascending aorta, after giving off its three branches, merges as a thinner vessel into the pulmonary artery. If the branch of the pulmonary artery which bends down to the descending aorta, and represents the Ductus arteriosus, be closed,—which, singularly enough, happens in all cases,—the descending aorta cut off from the pulmonary artery, approaches the ascending aorta so closely as to leave only a very narrow connecting link between them, viz., the thin vessel already described, merging into the pulmonary artery which represents the descending aorta.

2. *This portion of the vessel, from its narrowness, presents the conditions requisite for its obliteration and atrophy.* It becomes narrower, with increasing years, in relation to the ascending aorta and to the quantity of blood passing through that vessel, while, at the same time, not only the ascending aorta, but the branches given off from it are proportionally dilated. This dilatation soon extends over all the ramifications and their anastomoses, as, for instance, those of the internal mammary and the first intercostal with the remaining intercostal and the epigastric arteries. In proportion as the collateral circulation draws the blood more freely from the left side of the heart and from the isthmus between the arch and the descending aorta, the former becomes narrower, and is at length rendered useless, completely closed, and finally atrophied.

The heart is, in all these cases, more or less distinctly affected with active dilatation. This dilatation obviously depends at first on the narrow isthmus between the ascending and descending branches of the aorta, and, after the latter has become atrophied, on the inefficiency of the collateral circulation.

This contraction and closure of the aorta has been observed from the fourteenth to the ninety-second year. It occurs far more frequently in men than in women. There are fifteen or sixteen such cases on record.¹

¹ These cases, as already remarked, were collected by Barth (*Presse Médicale*, 1837,) and by Craigie, (*Edinburgh Med. and Surg. Jour.*, Oct. 1841.) Craigie enumerates ten cases, among which, according to Hasse's statement (*Path. Anatomie*, Bd. i, p. 91), one of the cases collected by Barth must be wanting. I unfortunately have not Barth's memoir by me at the present moment. He collected nine cases, which include Otto's case, (which Hasse must have missed in Barth's collection, as he specially enumerates it, although it is not given in Craigie's list,) Römer's case (*Oest. Jarhb.*, Bd. xx, St. 2), and the case observed by Craigie, and described in the above named memoir. These, together, make twelve cases, to which four others have been recently added—one case observed by M. Aug. Mercier in the year 1838 (which Craigie has overlooked), a case observed by Muriel in 1842, one described by Hamernik in 1843, and one case, also occurring in 1843, which is preserved in our Pathological Museum.

We think that a more detailed notice of the last named cases may contribute to the completion and elucidation of Barth's and Craigie's series.

The *thirteenth case*, observed by M. Aug. Mercier (*Bulletin de la Société Anatomique de Paris*, xiv. année, p. 158):

Contraction, with almost complete Obliteration, of the Thoracic Aorta.—Potier a shoemaker, aged 38 years, was received into La Charité on the 29th of March, 1838. He had been seized, in the October of the preceding year, with violent bleeding from the nose, which continued for three hours. This occurrence of epistaxis relieved him from attacks of giddiness to which he had been previously subject. Towards the month of January he suddenly experienced a sensation of cold and weakness (paralysis) in the right hand, which disabled him from using his knife. This sensation disappeared in about a week. From the 27th of February he had had an occasional cough; but it was not until the 27th of March that blood was expectorated, which increased very considerably on the 28th and 29th.

The patient experienced pain in the region of the apex of the heart, and forwards and backwards at about the same elevation, which prevented him from lying on his side. The beats of the heart were frequent, but without any peculiar sound; but at the summit of the arch of the aorta, a strong bellows sound, continued into the carotids, was heard in unison with the arterial pulse. The sound was almost equally loud at the lower angle of both scapulæ, where two or three of the intercostal arteries were observed to pulsate with violence. The pulse at the wrist was 140, very large and hard, but otherwise regular. There was no sound along the femoral arteries, which beat so faintly that they could scarcely be felt. An obstruction to the current of blood in the descending thoracic aorta was diagnosed.

On the 31st there was a violent pain at the top of the ninth dorsal vertebra, between the spinal column and the scapula. The symptoms of pleuro-pneumonia increased, notwithstanding energetic treatment; and the patient died on the 9th of April.

Autopsy.—The left pleural sac contained a coagulated and fluid exudation; the lung was in a state nearly approximating to pneumonia in its third stage.

The heart was large, and invested with pseudo-membranous coagula; the aorta,

Death is generally occasioned by the heart-disease and the anomalies to which it gives rise; in four cases it was owing to rupture, twice of the ascending aorta, once of the right ventricle, and once of the right auricle. Here, as is generally the

together with the arteries branching off from it, was dilated from its commencement to about a few lines below the origin of the left subclavian. In the middle of the free margin of one of the aortic valves, there was seated a whitish-red, and apparently old coagulum.

About five lines below the point of origin of the left subclavian, the aorta appeared to be almost entirely obliterated. The opening, which would only admit a blunt probe, was closed up with coagulated blood. It was linear in form, and surrounded by a posterior and an anterior lip, the latter of which projected far less than the other, causing the opening to approach nearer to the anterior than the posterior wall of the aorta. The tissues appeared to be normal at the contracted spot.

The Ductus arteriosus was obliterated, terminating in the concave portion of the aorta, about three fourths of a line above its contraction.

The contraction extended over a very inconsiderable space, and was sharply defined both at its commencement and its termination. Immediately below it, the calibre of the aorta scarcely varied perceptibly from its normal dimensions. The abdominal aorta, and the iliac and femoral arteries, were also only slightly smaller than usual. The pair of intercostal arteries, branching off above the contraction, were 2''' in diameter; the remainder gradually decreased in calibre to the fourth, which appeared to be normal.

Fourteenth case, described by William Muriel in the seventh volume of 'Guy's Hospital Reports' for 1842.

James Bert, a labourer, aged 25 years, of small stature, died on the 27th of July, 1842. Nine years previously, he had suffered from symptoms resembling those of an aneurism of one of the larger vessels of the chest. The symptoms gradually abated under the proper treatment, and, after a few months, he had so far recovered as to be able to work again; and was employed as a farm-servant uninterruptedly till the 20th of June, 1842. On that day, however, on lifting a heavy weight, he sprained himself. This accident gave rise to pain in the back and spasms, which were alleviated by opiates and counter-irritants applied over the spine. He lingered, however, until the 27th of July, when he died in a comatose state, which had been preceded with severe pain in the head.

On a post-mortem examination, the body was found to be somewhat emaciated, the chest deformed by the projection of the sternum, more especially towards the ensiform cartilage, and there was an inclination of the spine in the upper dorsal region towards the right side; the pericardium contained about three ounces of fluid; the heart was somewhat hypertrophied, with some dilatation of the ascending aorta, and of the vessels branching off from the aortic arch. At the point of union of the Ductus arteriosus, the aorta was *extremely contracted and almost obliterated*, whilst the superior intercostal arteries, more especially on the left side, were much dilated. There was no malformation of the heart. Opposite the contracted portion there was a hard tumour, about the size of a hen's egg, which was intimately connected with the aorta and the trachea, and formed by the bronchial glands. The left sides of the bodies of the third, fourth, and fifth dorsal vertebræ were partially

case with those heart-diseases which frequently continue unnoticed for a long time, and do not give rise to disturbances

destroyed in the region of the tumour; the lungs and the other viscera were healthy; the head and spine were not examined.

Fifteenth case, described by Dr. Jos. Hamernjk, of Prague (Oesterr. Wochenschrift, 1843, No. 10.)

N. N. Maurer, aged 42 years, who had always enjoyed good health, had been injured eighteen years previously by the upsetting of a carriage, which occasioned contusion and a dislocation of the scapular end of the clavicle. He was seized with pains in the feet and œdema; and stated that, although these symptoms disappeared in a few days, he suffered ever since the accident from palpitation of the heart and headache, which were always removed by spontaneous epistaxis. About ten days before his death, he was attacked with pneumonia of the right side, and died on the 13th of February, 1843.

Dr. Hamernjk saw him two days before death, and found the temperature of the body higher than usual, the pulse at the wrist 120, and tolerably large. The pulsations of the heart might be seen and felt between the sixth and seventh ribs, while at the same time the next two upper intercostal spaces sank inwards; the same happened with the first named intercostal space at the diastole, whilst the two intercostal spaces above bulged out. The resonance in the region of the heart was not strikingly diminished, but the sounds of the heart, and along the greater arterial trunks, were not distinct, excepting perhaps the second sound in the pulmonary artery and the aorta, which were strong and very clear. A clear blowing sound was perceptible over the whole surface of the cardiac region, somewhat after the systole. This sound was strongest at the left border of the sternum, from whence it diminished in clearness, although it might be heard at a considerable distance (from the dilated internal mammary artery, which was rough). There was pneumonia of nearly the whole of the right lung, of which only the upper part seemed free. A somewhat rough bellows sound was perceptible in the carotids and the subclavians, as well as in the other large arteries. This sound was rather clear, and loud and protracted at the back, to the left of the vertebral column, at the posterior extremity of the second rib, and might be heard over the whole length of the vertebral column. Bulgings of the compressed and pulsating arteries were to be seen over the whole surface of the back, running in a twisted manner, and more especially diffused on both sides of the vertebral column in the direction of the axilla. There was no œdema.

On opening the body, pneumonia of the right lung was discovered. The heart was somewhat large; the cavity of the left side, however, was small, although its walls were upwards of an inch in thickness; the valves were normal, as was also the pericardium. The above described rolls of pulsating bulgings along the vertebral column, were the dilated and attenuated branches of the transverse arteries of the neck and scapula, as well as of the subscapular artery.

Dr. Hamernjk himself only saw two separate portions of the body, which he describes:

1. *On the walls of the chest* the two internal mammary arteries were laid open; their calibre was enlarged to the thickness of the little finger; their coats were interspersed at various points, with some few uneven cartilaginous plates, more especially at the upper portion of the vessel.

The portion of the Aorta.—The arch of the aorta, as far as the left subclavian,

in the system until they have attained a certain limit, the patients continued perfectly well up to a certain period, when

was only about an inch and a half in length; it measured 7" in diameter, and its walls exhibited their normal thickness and elasticity. The left subclavian artery was 6½" in diameter, and was therefore nearly as large as the remaining portion of the arch of the aorta. There were scarcely 2" of the subclavian artery remaining in the preparation, and the outer wall was invested with some thin plates of bone, as was also the posterior wall of the descending portion of the aorta. About one inch below the point of origin of the subclavian from the aorta, the latter was suddenly contracted circularly, but more especially at the back, by a deep furrow, so that, with its walls included, it did not exceed 5" in diameter when measured from right to left. The contracted portion scarcely measured 4" from before backwards, and was therefore somewhat flattened. Above the confined or contracted portion, the aorta was swollen to about the size of a middling-sized hazel-nut, and ossified. At the point of contraction, a transverse wall was observed, having the form of a bi-concave lens, and about 1—1½" in thickness, which entirely closed the tube. Below the contracted portion, and about 2" deeper, the aorta began suddenly to dilate, and measured 12½" in diameter. This dilatation extended over a length of about an inch and a half. The aorta then again assumed its normal diameter; and about 1 inch above the diaphragm it was very slightly dilated. The intercostal arteries of the right side, more especially the second and seventh, were dilated; the former was at least double its normal width. Its walls were thin and collapsed.

The remains of two small shrivelled vessels, with contracted tubes, lay close together on the concave wall of the contracted spot, where each ended in a *cul de sac*. They corresponded to the opening of the Ductus Botalli. Dr. Hamernjk was not able, from the restrictions imposed on his use of the preparations, to discover whether this character depended on original division (duplicity) of the arterial passage, or whether it could be regarded as owing to acquired shrinking or puckering. There was no roughness or cartilagescence to be seen below the obliteration.

Dr. Hamernjk is of opinion that these appearances were due to original formation.

Sixteenth case, in our pathologico-anatomical Collection. Dr. Dlauhy, who conducted the post-mortem examination, has given me the following particulars:

Harzmann Ignaz, aged 27 years, a day labourer, suffered for some years before his death from slight erysipelas of the face, during one winter and a succeeding autumn. For more than a twelvemonth he had experienced considerable palpitation of the heart at night, after hard work. During the last three months this had been frequently associated with cough and expectoration of tough mucus, and with oppressed respiration. This condition grew rapidly worse; and for two months before his death, which occurred in the beginning of March, 1843, he had œdema of the feet.

Autopsy.—The body was of a robust make; there was œdema, more especially of the lower extremities; the abdomen was much distended and fluctuating.

The Sinuses of the Dura Mater were distended; the Pia Mater, together with the brain, abounded in blood.

The abdominal cavity contained about 20lbs. of clear serum, intermixed with scattered fibrinous flocculi. The liver was not much enlarged; its substance was

the symptoms of heart-disease were either gradually or suddenly manifested.

2. *Contraction and Obliteration in consequence of Disease of the Coats of the Vessel.*—To this class belong:

a. *Obliteration of the mouths of a vessel, occasioned by the excessive formation of a tissue, analogous to the lining membrane,*

distinctly separated into a yellow and a dark reddish tissue (nutmeg liver); its peritoneal investment was thickened, and in some spots had a tendinous appearance. The gall-bladder contained tough, dark brown bile. The spleen was dense, of a dark reddish-brown colour, and tolerably large. The kidneys large, and very tough. Ramifications of veins, much filled with blood, were observed on the ileum.

Both lungs were, for the most part, attached by cellular adhesions to the costal wall; the cavities of the pleural sacs contained about a pound of serum; both lungs were puffy, and oedematous; in each of the lower lobes there was a spot of the size of a pomegranate, in addition to several smaller ones, of a blackish-red colour and fragile,—a hæmorrhagic infarctus. The mucous membrane of the trachea and the bronchi was bluish red, and loosened; and the bronchial ramifications were filled with a thick, yellowish, puriform mucus.

There were about two ounces of clear serum in the pericardium. The heart was more than twice the normal size, and invested with numerous milk-spots; the muscular substance was tough throughout, and of a reddish-brown colour. The left ventricle was much dilated, and its wall was about an inch in thickness; and the right ventricle and the left auricle were dilated and hypertrophied. The valves were normal; the Foramen ovale was closed; the Venæ cavæ, the intercostal veins, the jugular veins, &c., were dilated and swelled.

The Aorta.—The preparation consisting of a part of the ascending aorta, the arch, and a portion of the descending aorta, presented the following appearances:

The ascending arch of the aorta (regarded as the vascular trunk designed to supply the head and the upper extremities) was unusually extended downwards; after giving off the arteria innominata, it diminished so much that its diameter did not exceed 3" at the point where the left subclavian was given off, which formed, as it were, a continuance of it, and was of equal calibre with it. Above the valves its diameter was 11". From this point it was deflected rapidly, and almost angularly, as a vessel of about 11" in length, and not more than 3" in diameter; its lower extremity corresponding to the depression of the obliterated Ductus arteriosus, was contracted and already undergoing obliteration, and was cut off from the descending aorta by a deep furrow.

At this spot the calibre of the artery scarcely measured one line; the passage, which only admitted a thin probe, was obstructed in the direction of the descending aorta by a small plate of white, opaque deposit. The descending aorta varied from 8 to 9 lines in diameter.—The deposit was very considerable, opaque, and partially ossified, in the ascending aorta; the walls were rigid. The descending portion exhibited only a few plates of an opaque deposit. The arteria innominata was about 8 or 6 lines, the left carotid about 2 inches, and the left subclavian, as we have already remarked, about 5" in diameter. The ends of the intercostal arteries branching off from the descending aorta, more especially the uppermost ones, were considerably thickened.

within a trunk.—This condition, which is followed by atrophy of the vessel itself, is a mode of obliteration of the arteries which has not been much regarded, and one whose nature is not known. It is the result of the excessive process of deposition in one of the trunks of the vessel and in the vicinity of the mouth, as has already been described at p. 262. The mouth continues to become narrower, until it is finally closed by the last layer deposited around it, whilst it diminishes by the fusion of the mass around the circumference. After this the mouth very frequently appears as if it were closed by a membrane stretched across it. When the vessel has become shrivelled and wasted, the closed mouth presents a cicatrix-like puckered appearance, or has wholly disappeared. Above the closed mouth the blood carried by the collateral circulation into the vessel coagulates over various extents of surface, until its coagulation is prevented by the circulation established by an anastomosing process. The artery shrivels and becomes atrophied above this clot or plug.

As this process of deposition must be very highly developed in order to produce such occlusions, and as it results in dilatation of the diseased artery, we are able to explain the appearance of these contractions and final obliterations of the mouths of the vessels, more especially upon the branches going off from aneurismal vessels. (See p. 286.) We have already alluded generally to the importance of this obliteration, which is, indeed, self-evident; but it exhibits special interest in some individual cases, among which we may notice the following:

1. The contractions and obliterations of the branches of vessels passing from the arch of the aorta.
2. The contractions and obliterations of the coronary arteries of the heart.

Neither of these is by any means a phenomenon of rare occurrence.

b. The contraction and final impermeability of an artery in consequence of excessive deposition—of its ossification—or of the deposition of fibrinous vegetations on the rough inner surface of the vessel, and their cretification. This may be especially observed where the process of ossification is much diffused on the smaller branches of the femoral arteries; many cases of

senile gangrene are based on this impermeability of the arteries, which, however, is seldom observed in vessels of considerable calibre.

3. *Occlusion of the Arteries.* To this class belongs the occlusion of the vessel arising from different varieties of coagulation of blood.

a. *Occlusion of an inflamed artery.*—According to our definition of arteritis, this condition can only affect arteries in which the circular fibrous coat is only subordinately developed. (See p. 253.)

b. *Occlusion of an artery arising from a coagulation of the blood, depending upon an internal cause, such as a blood-disease.*—To this class belong Velpeau's case of closure of the aorta, from the third lumbar vertebra downwards, with a part of the iliac artery, owing to a coagulation of a cancerous character in an individual exhibiting cancerous cachexia, and undoubtedly also the cases of occlusion of the thoracic and abdominal aorta observed by Schlesinger and Barth. Occlusion arising from arteritis, and especially the form above described, is very rare when compared with the frequency of occlusion of the veins.

4. *Contraction and obliteration arising from persistent pressure on the artery.*—Such a continued pressure may be exerted by different tumours, as goitres, encysted tumours, cancerous products, and aneurisms of neighbouring arteries. Complete obliteration is very rarely induced by these causes; at any rate, in the larger arteries. The vessel becomes obliterated at the spot exposed to pressure in consequence of the coalescence of the lining membrane of the vessel; above this point the occlusion is effected by means of a plug reaching to the nearest branch, and beyond this the vessel is finally obliterated in the same manner as after tying the artery, as we shall have occasion to revert to in the sequel.

Besides these different modes of contraction, occlusion, and obliteration, we further noticed the following conditions when treating of aneurisms. (See p. 286.)

a. *An impermeability of the mouths of the branches passing from an aneurismal vessel in consequence of their contraction into fissure-like openings.*

b. *An impermeability of the mouths of these vessels, induced*

under certain conditions already indicated at the above page, *by the fibrinous layers filling the aneurismal sac*, and the shrinking and obliteration of the vessel consequent upon it.

We shall consider the establishment of the circulation consequent on the obliteration of an artery when we treat of the healing of cut arteries, and the process of obliteration that follows the tying of an artery.

5. *Mechanical Separations of Continuity.*—To these belong *lacerations and wounds of the arteries produced by cuts, thrusts, or gun-shot wounds.*

Spontaneous lacerations are the most important of any, especially those of the trunk of the aorta. To this class belong *lacerations of the large arteries arising from violent concussions or shocks*, viz., in consequence of a contusion, (as, for instance, by a spent ball,) striking a circumscribed portion of the vessel. No special interest attaches itself to those lacerations of the arteries which arise from excessive extension; such as, for instance, in the arteries of the extremities from dislocations, and which may be associated with extensive lacerations of the soft parts, and destruction of the bones.

Wounds of the arteries inflicted by cuts, thrusts, and shots, derive importance from the conditions of *false* and *varicose aneurism*, in which they frequently result.

A. *On the Lacerations of the larger Arteries.—Dissecting Aneurism.*

Lacerations of the larger arteries, arising from *traumatic influences*, as from concussions and contusions of the body, are only interesting in a scientific point of view, when the different mechanical modes of laceration affect differently the separate arterial coats, and when they resemble certain spontaneous lacerations. Such is the case when the laceration implicates the two inner coats of the vessel (the lining and yellow membrane), while the cellular sheath of the vessel is in a state of integrity, or when the separation of its continuity does not correspond in extent, form, and direction with this laceration.

In this respect we must regard with special interest the *læsiones continui*, which are owing to some influence limited to a circumscribed portion of an artery, or to a loosening of the cellular sheath and a laceration of both its inner membranes

owing to the same influence. They present the greatest similarity with that spontaneous laceration which is termed *dissecting aneurism*.

*Spontaneous lacerations*¹ may be classified under the following heads.

1. *The laceration depends upon a delicacy of construction of the whole arterial wall, and on the generally simultaneous narrowness (insufficiency of calibre) of the vessel; or on congestion, or excessive expansion of the mass of the blood.* We have observed several cases belonging to this class.

2. *The laceration depends upon a diseased condition of the texture of the coats of the arteries.*

The cases belonging to this class form two distinct series.

a. *In those of the first series, the læsio continui consists in a detachment of the cellular sheath from the tube of the vessel, and of a laceration of the middle and lining coats of the vessel within the detached cellular sheath.* The question here arises, which of the two is the primary, and at the same time the controlling cause?

Experiments prove that it is by means of the cellular sheath, more especially of its elastic longitudinal stratum, that the artery is able to resist any violent lacerating action, and to sustain the force of the blood-wave when the texture of the inner layers, particularly of the yellow coat, is in a state of integrity.

In the above cases, the alteration of texture consists essentially in a chronic inflammation of the cellular sheath, which causes it to be more easily detached. The cellular sheath is here loosened, over various extents of surface, from the tube of the vessel, either alone or with an adhering layer of the yellow membrane, which is generally torn transversely, and only very seldom longitudinally to the vessel. The yellow coat is very brittle in the cases to which we refer, and where this condition was certainly the result of advanced age, this membrane admitted readily of being separated. The lining membrane was for the most part diseased, although only in a moderate degree, exhibiting a deposit which was partially ossified.—By way of elucidation we will give a case borrowed from the memoir before referred to.

A. G. v. P.—, aged 52 years, a widow, fell to the ground in

¹ Oesterr. Med. Jahrb., Bd. xvi, St. 1.

the street, on taking a quiet walk after dinner, towards evening, on the 18th of February, 1833. After being bled, she was carried to the hospital. She was brought senseless and pulseless. She vomited twice, and after momentary recurrences of consciousness, died on the following morning, after long-continued and profound syncope.

Autopsy.—The body was of moderate size and thin.

The walls of the cranium were 3–4" in thickness, and compact; on the left parietal bone, above the semi-circular line, a compact exostosis was discovered, about the size of half a walnut; the inner cerebral membranes were infiltrated. Some of the arteries on the base of the brain were partially ossified.

The lungs, with the exception of the swollen anterior margins of their upper lobes, were of a dark red colour, rich in blood, and œdematous at different points. The left pleural cavity contained 1lb., and the right cavity about 4 oz. of pale reddish sanguineo-serous fluid.

There was considerable extravasation of coagulated blood in the posterior mediastinum round the aorta and the œsophagus, more especially, however, round the pulmonary vessels and the branches of the trachea, towards the roots of the lungs; the pericardium contained upwards of a pound of coagulated and fluid blood.

The heart was somewhat larger than usual, nearly of a round form; the left ventricle and the Conus arteriosus were very large, and the walls of all the cavities were of normal thickness. The right side of the heart was covered with a considerable layer of fat; the ramifications of both the coronary arteries were, for the most part, ossified. The substance of the heart was pale and friable.

Aorta.—The ascending aorta, like the pulmonary artery, was very wide; the valves of the former were thickened at their insertion and their nodules, and were partially ossified. The cellular sheath (the elastic and cellular coats) of the aorta was loosened throughout its entire length round the ascending portion, at its arch and on the whole of the thoracic and abdominal aorta, over full a third of its circumference, where there adhered to it either a thin layer or partially exfoliated thicker portions of the yellow coat. This condition extended upwards over the arteria innominata to the common carotid, the right

subclavian and its larger branches, and downwards over a portion of both the iliac arteries; in the former the cellular sheath was entirely separated over the whole circumference of the artery, while in the latter it was only partially loosened, or admitted of being easily detached all round, together with the external layer of the middle coat. The same condition was observed in most of the small and large branches of the thoracic and abdominal aorta for a considerable extent of surface from their points of departure. The cellular sheath was of a bluish red colour, infiltrated with blood at many parts of its detachment, and very thick; at those points, however, at which a layer of the yellow coat still adhered to it, and where the two were not entirely separated, it was paler and less thick from a deficiency in the suffused blood, and was intersected by a highly developed net-work of vessels. The free space between this and the yellow coat of the vessel was filled with a considerable quantity of coagulated and fluid blood.

Within the cellular sheath, which was detached, as we have already seen, from the ascending aorta, the yellow and lining membranes were torn transversely over an extent of an inch and three-quarters above the valves, so that there remained only a spiral strip of their posterior wall, (about two lines and a half in breadth, and equal in length to about half the circumference of the aorta,) which connected together the two extremities of the rent, and was raised along the concavity of the trunk of the aorta from its original horizontal position, in consequence of the displacement of the upper extremity of the rent, which we shall now proceed to notice.

While the lower extremity of the rent was turned upwards with an almost circular opening, in consequence of the exposed transversal rent, the upper one was almost entirely enclosed by the convex wall and driven into the cavity of the arch of the aorta as far as the left subclavian, the entrance of which was even obstructed by a conically rolled portion of the tube of the aorta, so that a communication was opened from the subclavian into the inserted vessel, and through this into the cavity of the cellular sheath.

In this manner, both the extremities of the rent were from about an inch and a half to an inch and three quarters from each other, while within the almost saccularly expanded cellular

sheath at this spot, as well as over the whole extent of the aorta and the branches already referred to, the space between it and the yellow coat of the artery was completely filled with coagulated and fluid blood. This accumulation of blood had compressed the aorta and the cœliac axis at different points, and completely detached from their origin several small branches of the aorta and a large branch of the renal artery on the left side.

The blood had been further extravasated from this space into the pericardium and the mediastinum, in the following manner. The cellular sheath of the ascending portion of the aorta was torn outwards and backwards along the descending Vena cava, near its opening into the auricle, in a longitudinal direction, together with the portion of the pericardium by which it was invested. This sheath was also considerably attenuated at several points along the descending aorta, where it readily admitted of being torn.

The yellow coat of the artery could be easily peeled off in all parts, but more particularly at the aorta itself; it was also very brittle. Several small bony plates were observed in the lining membrane of the arch of the aorta.

The intestines were pale throughout, although this pallor was especially perceptible at some circumscribed portions of the ileum where the mucous membrane was perceptibly attenuated, and had even wholly disappeared.

When we consider the appearances here presented, with a view of ascertaining the relation of the different coats of the artery in their physiological and pathological condition, we arrive at the following theory, viz., *that a detachment of the cellular sheath occurs spontaneously at a certain stage of its disease, giving rise at the same time to laceration of the two inner coats.* These coats are usually torn transversely along the course of the yellow fibres, in consequence of the artery being deprived, at the moment when the cellular sheath is detached, of the support which had limited its further expansion and stretching. Such a laceration is also the more readily effected, when the two inner coats, notwithstanding the integrity of their texture, are unable to resist this expansion and tension from having become soft and brittle, owing, as is commonly the case, to advanced age, or to the dilatation of the vessel which is observed in all such cases.

The detachment of the cellular sheath must, therefore, constitute the primary agent or cause, while the laceration of the inner coats is the consecutive effect of this condition.

It is, however, probable that this may admit of a different explanation. Thus, for instance, we are unable to apply this theory when, in addition to the cellular sheath, an adhering layer of the yellow coat is loosened with it at the spot of the laceration, and when, therefore, the rent itself affects only the inner layer of this coat (with the lining membrane of the vessel). Another theory suggests itself when we consider that dilatation of the vessel is present in all cases. *This dilatation depends, in all probability, upon the paralysis of the elastic layer, owing to a chronic inflammation of the cellular sheath; and the laceration of the yellow coat of the artery might therefore be the final result of the greatest dilatation it was capable of resisting without any considerable disturbance of texture of the whole arterial wall.* Laceration will, moreover, be the more readily effected in proportion to the brittleness of the yellow coat dependent on the advanced age of the patient.

In accordance with this view, the rent in the lining and yellow coats must be the primary occurrence, and the loosening of the cellular sheath, either with or without an adhering layer of the yellow coat, must be regarded as a secondary result owing to the forcible escape of blood from the rent. The following conditions appear from our observations to be worthy of notice as controlling causes:

The heart is hypertrophied in all cases, and its left ventricle is in a state of active dilatation. In most cases the laceration is effected without any special excitement of the heart's action, so that the occurrence must be regarded as the final result of the diseased condition of the vessel.

The integrity of the detached cellular sheath, that is to say, the hinderance thus opposed to the free extravasation of the blood, occasionally postpones the fatal termination for a few hours; in Laennec's case death was delayed for four days.

The cellular sheath is generally lacerated in consequence of its distension by extravasated blood, usually in the vicinity of the lining membrane of the artery, but occasionally, however, one or more spots remote from that rent.

These lacerations are generally *transverse*, and only rarely take a longitudinal direction.

Lacerations are much more frequent in the ascending aorta, at a short distance above the valves, than in the thoracic aorta.

They generally occur in persons of advanced age.

This form of lacerations belongs to the class which has been repeatedly investigated by English pathologists, who have applied to them the inappropriate designations of *dissecting aneurisms*, or of *anomalous* or *interstitial aneurisms*. These observers have not hitherto given a feasible explanation of this process, and they appear to have overlooked the conditions that induce such diseases of texture.

b. The cases belonging to the second series differ in every respect from those of the first. *These are lacerations of an artery exhibiting a profoundly diseased condition of the texture of the whole wall—somewhat in the manner of the so-called dissecting aneurism, that is to say, with detachment of the cellular sheath, but this is here always effected by the violent action of the blood extravasated from the rent, and therefore constitutes a secondary occurrence.*

A cause predisposing to these lacerations is afforded by a high degree of the disease which we have described at p. 262, and to which we have referred as the cause of origin of aneurism. The cellular sheath of the vessel here firmly coalesces with the yellow coat of the artery, in consequence of a process of chronic inflammation by which its tissue becomes thickened, callous, and condensed.

The inner coats of the dilated artery are lacerated in consequence of their morbid brittleness within the closely adhering, thickened, callous, resistant, cellular sheath, which is here violently detached by the blood, but never over an extended surface, as in the cases belonging to the first series.

The cases belonging to this class are generally *longitudinal lacerations*, in which the fibres of the yellow coat of the artery are actually torn asunder. Transverse lacerations occur only as exceptions to the rule. When laceration takes place after a very considerable degeneration, with unequal disease of the arterial coats, the rents are irregular and curved. The following case may serve as an illustration of these appearances.

On the 6th of March, 1834, a post-mortem examination was

made of the body of a woman, aged 50 years, who had died suddenly two nights before. The autopsy showed the body to be robust, and in tolerably good condition. Both arms bore marks of repeated venesection.

There was a greyish white foam, collected in the trachea.

The lungs were of a dark-red colour, very full of blood, and œdematous, excepting in the right lower lobe.

There were two pounds of coagulated and fluid blood in the pericardium. The heart was half as large again as usual, fat, and flabby in the left ventricle, and the Conus arteriosus of the right ventricle was dilated. The auricles and trunks of the vessels contained coagulated and fluid blood.

The ascending aorta and its arch were considerably dilated; their inner surface was uneven, and covered at some parts with a white, opaque, cartilaginous, and smooth deposit, and at other parts with a light-coloured, wrinkled deposit of considerable thickness; the mouths of the three branches of the arch of the aorta were contracted. About an inch and a half above the semilunar valves on the concave wall of the ascending aorta there was a jagged, rectangular rent in the diseased inner and middle (yellow) coats of the artery. The longest direction of the rent measured one inch and five lines, and ascended into the arch of the aorta, while its other side (which was only half the length of the former), extended along the posterior wall of the aorta. A rectangular lobule, composed of a portion of the inner and of half the thickness of the middle coat, had been exfoliated from the above described right angle, and from this point the ascending aorta had lost the cellular sheath, together with the external layer of the middle coat, except at a mere narrow strip on its concave surface. The space between these two laminae was filled with coagulated blood. The external lamina had burst into the pericardial cavity backwards, behind the descending Vena cava, longitudinally and downwards, over a surface extending more than half an inch, together with the contiguous lamina of the pericardium.

The cellular sheath of the aorta, was unusually thick, although at the same time of a callous condensation, and intimately connected with the yellow coat. It was of unusual thickness, nearly 1" at the arteria innominata, and more especially at the right subclavian, and was converted into a whitish,

very dense and tough, lardaceo-fibrous stratum, and fused as it were into the yellow coat. It was less thick at the left carotid and the subclavian, although it presented a similar character.

On examining the abdominal cavity, the gall-bladder was found to contain a concretion, about the size of a nutmeg; and the fundus uteri was filled with a fibroid growth, equal in size to a child's head, and attached by a thick pedicle.

These Lacerations, like the diseases in which they originate, generally occur in advanced periods of life. They also usually affect the ascending aorta, which may be explained by the circumstance, that this vessel is, in most cases, especially diseased, while it is at the same time exposed to the force of the blood-wave propelled from the heart.

The heart, as may be conjectured from the observations already made, is subject, in these cases, to dilatation and hypertrophy, more especially of the left ventricle.

Among the aneurismal forms, especially allied to these cases of the second class, we must reckon *lacerations of the smaller, diseased arteries, having rigid membranes and having become brittle, which are either frequently spontaneous, or the result of wholly inexplicable conditions, such for instance as we especially see in apoplexy (cerebral hæmorrhage).*

3. *Finally, this laceration may depend upon the removal of the supports of an artery, in consequence of an ulcerous process, and upon a loosening and softening of its texture, arising from its coats becoming infiltrated with the ulcerous secretion surrounding them.* This form of laceration more particularly affects delicately constructed arteries of inferior calibre, as, for instance, the branches of the pulmonary artery in the walls of tuberculous pulmonary caverns. In some few cases the laceration is preceded by a lateral (aneurismal) enlargement of the vessel towards the cavern. (See p. 261.)

B. *On Incised, Penetrating, and Gunshot Wounds of the Arteries.*

Such injuries of the artery as are inflicted by sharp-pointed instruments, even where it is only opened at the side, and shot-wounds which merely remove a small portion of the wall of an artery, are, as is well known, extremely dangerous; for

they usually give rise to the so-called *false aneurism*, and, under certain conditions, to *varicose aneurism*, which we shall soon consider in detail. It is true that penetrating and incised wounds of an artery may heal under favorable conditions, as we see in cases where the temporal artery has been opened, and, as Amusat has recently shown, by observations at the bedside and by experiments on animals, in the same manner as similarly injured veins. But, as in man, injuries are often inflicted on the arteries under circumstances which exclude the concurrence of these favorable conditions, such wounds do not commonly heal; in gun-shot-wounds of the artery more especially, a cure is never effected by the adhesion of the margin of the wound, but, as an ordinary consequence, we generally have the so-called *false aneurism*.

On False Aneurism.—When an artery of one of the extremities has been injured in any of the above ways, the blood is effused into the surrounding cellular tissue, forming an extravasation, if unable to escape from the outer wound. The blood is then accumulated in a cavity formed by the laceration of the tissue, the structures around it being suffused and infiltrated with blood. This constitutes *diffuse false aneurism*, or, according to Foubert, *primary false aneurism*. When considerably diffused it in general terminates fatally in inflammation degenerating into gangrenous disintegration, associated with symptoms of paralysis, and in continuous external hæmorrhage. It is only in rarer cases, and when the aneurism is less diffused, that it can heal by the artery becoming obliterated during the ichorous process, through arteritis, and by the drying up of the ulcerous process, after expulsion of the extravasation and of the tissue destroyed by it.

When the neighbourhood of the extravasation becomes the seat of an inflammatory process, (reaction,) tending to condensation (sclerosis) and hypertrophy of the tissue, the cavity containing the extravasation acquires a true wall and definite limits, and becomes converted into a sac, seated upon and surrounding the artery, and into which the arterial wound opens. A lining membrane may be formed upon its inner surface, and the sac may then present such similarity with a mixed *aneurism* as to render its anatomical diagnosis extremely difficult. In this condition, the collective appearances represent

what is commonly known as *false circumscribed*, or Foubert's *consecutive false aneurism*.

It is obvious that these conditions do not originally merit the designation of aneurism, whilst the consecutive condition of a *false circumscribed aneurism* presents appearances which give it in every respect the significance of an aneurism. This form of aneurism, which commonly attains a very large size, as, for instance, in the popliteal space, generally, as is the case with large aneurisms, terminates fatally when left to run its course.

On varicose aneurism, Aneurysma spurium varicosum, Varix aneurysmaticus, A. per anastomosin (W. Hunter), *A. per transfusionem* (Dupuytren).

This aneurism consists in the communication of an artery with a neighbouring vein, effected by means of an aperture in the artery corresponding to one in the vein. This communication may be *direct* or *indirect*, and further may be the result of injury, or may occur *spontaneously*.

Varicose aneurism is most commonly produced by some injury which simultaneously affects the contiguous walls of an artery and of a vein; such, especially, as penetrating wounds or injuries from small shot, and where it arises from incised wounds, it is in general owing to venesection in which both walls of the vein have been cut through, and the lancet has penetrated through the wall of the artery below it. The latter mode of injury is, moreover, the most frequent cause of *varicose aneurism*, and consequently the bend of the elbow is the most common seat of this aneurismal formation. Moreover, neighbouring arteries and veins may be so much injured by splinters of bone that the arterial blood may enter a vein. The same result has also been effected by violent contusions.

The most common form of traumatic varicose aneurism is that occurring after venesection, and seated between the brachial artery and the median vein, or when the brachial artery divides higher up, between the radial or ulnar artery and the median, cephalic, or basilic vein. The same form of aneurism has also been observed in the brachial artery in the upper arm, in the subclavian, in the carotid with the jugular vein, in the femoral, popliteal, temporal, and other arteries.

Spontaneous varicose aneurism is the opening or rupture of

an aneurism into a vein that has coalesced with it. Cases of this kind have been noticed by myself and many foreign observers in the femoral artery, in the abdominal aorta with the Vena cava inferior, and in the ascending aorta with the Vena cava superior.

The communication established between the artery and the vein is, as we have already remarked, either *direct* or *indirect*. The former is frequently observed at the elbow, as a consequence of venesection; thus, for instance, whilst the outer wound of the vein is cicatrising under a bandage and compress, which prevent the formation of an extravasation into the cellular tissue, the two vessels coalesce together, more especially in the immediate vicinity of the openings of the wounds, by which means a direct communication is formed between them. The vein at the spot presents the appearance of a swelling or roundish expansion, which commonly increases to about the size of a hazel nut or half a walnut, but, in some rare cases, attains an extraordinary volume. (Hodgson, Larrey.) The direct communication of the two vessels, and the dilatation of the vein at the corresponding point, constitute what is known as *aneurysmal varix*.

Such a communication between the artery and the vein is always present in spontaneous varicose aneurism.

The *indirect mode of communication* is effected in the following manner by the presence of a false aneurism. In those cases in which a sufficiently strong compress has not been applied immediately after the injury, and where two vessels have been injured, which are not in immediate contact, or are not fixed in one common layer of cellular substance, or where finally the wounds in each do not originally correspond together, or where, after the injury has been inflicted, the vessels have been displaced or separated,—an extravasation into the cellular tissue is formed, which, in the last-named cases, pushes the vein aside from the artery, and thus prevents the establishment of a direct communication between the two.

This extravasation,—a false diffused aneurism,—is now reduced to a false circumscribed aneurism, the cavity of which forms the medium of communication between the artery and the vein. This false aneurism varies in size, but it commonly is not larger than a walnut or a hen's egg. It generally forms a

more considerable and a tougher swelling than that which occurs in a direct communication, and hence Scarpa was led to distinguish this condition from aneurismal varix by the designation of *varicose aneurism*.

The aneurismal sac presents many differences in reference to the extent to which it lies on the artery, and especially in relation to its opening.

The size of the openings into the two vessels also varies very considerably, and depends, like their form, on the size of the original wound, on the instruments by which the injury was inflicted, &c. The opening in the artery in general permanently retains its original size and form, whilst that in the vein undoubtedly experiences many alterations like the aneurismal sac itself.

The openings in the vessels, as well as the inner surface of the aneurismal sac, are invested with a lining membrane of recent formation, which continuing into the two vessels, gradually loses itself, and merges, more especially in the case of the vein, into the deposit formed upon the inner surface of that vessel. In consequence of this, the openings in the vessels have a smooth and healed appearance.

In the so-called aneurismal varix and in spontaneous varicose aneurism, the communication is effected by means of a simple opening, through the coalescing adjacent walls of both vessels, which acquires a smooth and healed appearance in consequence of being invested with a membrane of recent formation, and thus loses its original character of a rent or rupture.

All observers are unanimously of opinion that the arterial blood principally, if not exclusively, passes into the vein, in both these modes of communication, the direct as well as the indirect. Breschet thinks, that in an operation for a varicose aneurism he also saw the venous blood pass into the artery through the aneurismal sac, during the diastole of the latter. To this circumstance, which may indeed occur in some cases, he refers the dilatation of the arteries below the aneurism, together with the attenuation of the arterial walls, in consequence of their contact with venous blood,—an explanation that has been given by no other observer.

The character of the vessels above and below the place of

communication, as indicated by all observers, and as I have repeatedly had occasion to notice, corroborates the existence of a very preponderating current of arterial blood towards the vein. The vein becomes first dilated below the point of communication, and then finally spreads beyond and above it. This dilatation is associated with an enlargement of the valves. The walls of the veins become thicker and more rigid, principally in consequence of the formation of new layers of lining membrane, (See p. 271;) they at length acquire an appearance similar to that of the arteries. Below the communication the artery is contracted in consequence of the blood being drawn away towards the vein; and its walls then become thinner, relaxed, and more similar to the veins in consequence of the diminished energy of their function, corresponding to the extent to which the blood is drawn away from it. The artery becomes dilated above the communication, in consequence of the obstacle which the venous blood opposes to the entrance of arterial blood into the vein.

The limb below a varicose aneurism is frequently swollen; it also presents a cyanotic colour, its cellular tissue is infiltrated and hypertrophied, and the general investment is the seat of repeated erysipelatous inflammations, excessive epidermal formation, &c.

The sequelæ of spontaneous varicose aneurism between the trunks of the aorta and of the Vena cava are obvious, but they are often indistinctly manifested in consequence of their being masked by the results of the aneurismal affection of the trunk of the artery, and by the simultaneous presence of heart-disease.

The process of healing and obliteration after arteries have been cut through or tied.—An artery, on being cut through, is immediately retracted in its sheath,—at any rate, as far as the next lateral branch, if of considerable size, while it at the same time becomes gradually contracted. The blood pours outwards in a greater or less quantity, according to the extent and position of the external wound, or it is effused into the cellular tissue. Independently of the aid afforded by art, the exhaustion itself exerts a favorable action on the coagulation of the blood effused around the artery. The coagulum within the canal of the arterial sheath, which is produced by the retraction

of the vessel, forms the *outer plug*,—the most essential and the actual means of arresting the hæmorrhage. In addition to this plug, another—the *inner plug*, is gradually formed within the artery itself, by the coagulation of the blood which is arrested in the stump by the first plug, unless a considerable lateral branch is given off in the immediate neighbourhood.

By these means the necessary conditions are obtained for arresting the hæmorrhage; and the cure—closure—is then effected by the adhesive inflammation of the cellular sheath of the vessel on the margin of the wound, and by the obliteration and final atrophy of the whole stump of the artery, as far as the next collateral branch, in the same manner as we observe after ligature, torsion, &c., and which we will now proceed to consider.

The whole healing process of arteries that have been cut through, has been fully elucidated by the invention of the ligature, and by the numerous investigations in reference to this process, as shown by experiments on animals. The labours of Stilling have thrown the greatest light on this subject in modern times; and we purpose in the following remarks to borrow from them the most important facts, which we will incorporate with our own views regarding individual points of the whole process, as obtained from investigations on the human subject. We would here briefly remark that the alterations resulting from ligature, &c., more especially the formation of thrombus, are more evident at the cardiac than at the peripheral end of the affected artery, and that the following remarks more especially apply to the alterations effected at the former of these points.

After the application of the ligature or torsion, the terminations of the cut artery, as has been already observed, retract. This gradual contraction which affects the artery as far as the next lateral branch, probably depends, at first, upon the irritation set up by the ligature, and subsequently, on the decrease in the quantity of blood entering this portion of the artery, in consequence of its abstraction towards the dilated branches, and on the diminished impulse. By this contraction of the fibres of the yellow coat the lining membrane of the vessel is wrinkled into delicate longitudinal folds; whilst the whole

of the wall of the vessel is puckered into more considerable plaits at the spot where the ligature has been placed.

The ligature or torsion takes the place of the external plug, causing a stoppage of the blood in the vessel between it and the first lateral branch. By being arrested, it forms a conical coagulum, that is to say, in the words of Stilling, "the arrested blood forms a conical mass, whose apex is near the first lateral branch, and whose base is seated on the extremity of the vessel, and is contained within a funnel of blood in partial motion. The larger opening of the funnel, where its walls are sharply cut and very thin, is in the neighbourhood of the base of this cone, or just above the extremity of the vessel, whilst its smaller (blind) opening whose walls are constantly increasing in thickness, until they finally unite, lies near the first lateral branch, in the axis of the vessel or the middle of its cavity." This conical coagulum constitutes an inner plug, which is commonly designated a *Thrombus*.

The thrombus begins to be formed at the termination of the vessel, and from thence extends onwards in its axis to the point where the first lateral branch is given off; it possesses a conical shape from its commencement, and not being very thick, it does not entirely fill up the tube of the stump of the vessel, but simply projects into it with its base or middle, at which parts it is usually of a dark, blackish red colour, and of very inconsiderable consistence. The apex, however, is white, more dense and hard, and resembles coagulated fibrin. It occasionally acquires a more fusiform shape by the addition of supplementary new layers, consisting in such cases of concentric superficial strata in addition to the original coagulum in the centre. It occasionally lies free in the stump of the artery, but more commonly it adheres, although at first loosely, by its base. It is subsequently invested with an albuminous moisture, by means of which it adheres loosely to the wall of the artery, although its apex remains free. This adhesion is frequently effected by means of filamentous bridge-like attachments.

The inflammation, which is set up around the injured vessel, also implicates its cellular sheath. Plastic lymph exudes into the tissue of all the structures and into the cellular sheath of the vessel. We find also that a process of adhesive inflammation affects the spot at which the ligature is applied, and where

the different folds of the lining membrane of the vessel come in contact with each other; and that there is an exudation of coagulable lymph, which causes a slight adhesion of the walls of the vessel, and of the thrombus at its base. The inflammation in the terminal part of the vessel is owing, not only to the irritation established by the operation, but also to the thrombus, which acts here as a foreign body; exudation being effused between the coats of the vessel as well as also on the free surface of the lining membrane.

The thrombus is always formed gradually; but in some cases it is found to be forming within half-an-hour or an hour after the operation, while, in other cases, there is no trace of it at the end of several hours. It is generally completely developed within twelve or eighteen hours after the closure of the vessel; it is more rapidly formed in small than in large vessels.

The further alterations include the metamorphosis of the developed thrombus, its coalescence with the wall of the vessel, and the final obliteration and atrophy of the artery.

The thrombus occasionally exhibits light coloured spots, both on its surface and in its interior towards its apex. Stilling observed fibrous or thread-like stripes on these spots, which he convinced himself by a lens were vessels.

In the course of time the thrombus acquires a porous structure, and becomes spongy and cavernous. Stilling found, in several experiments, that in addition to the numerous canals which he injected, and which traversed the thrombus in different directions, there was a central longitudinal canal opening into the cavity of the vessel. The periphery was especially injected in the more recent thrombi, whilst in those of older formation, the injection advanced more towards the centre or the axis. Where this so-called *vascularisation* was present, the thrombus was always of a paler, flesh-like, faint rose-red colour, turning to yellow, and finally to white, whilst the consistence was proportionally more considerable.—This metamorphosis of the thrombus is succeeded by its *regressive formation*.

In the meanwhile the thrombus becomes intimately adherent by its base, and very frequently by the whole of its body to the wall of the vessel,—in smaller vessels on the second or third day, and in larger ones on the fifth or sixth

day; the former adhesion has now been converted into a firm coalescence. The greater part of the apex of the thrombus commonly, however, remains free during the period of its greatest vascularity.

The regressive formation of the thrombus consists in the diminution of the number of the so-called vessels within it, in its increased pallor and density, and in the fact, that "the whole mass of the thrombus still remaining at this period, merges, as it were, into the mass of the stump of the artery, forming with it one body." (Stillings.)

This portion of the vessel gradually loses its proper texture; the exudation effused into the coats of the vessel becomes in part resorbed, and is in part metamorphosed into a cellular or fibroid tissue; the walls of the vessel gradually close around the shrivelling thrombus, and become obliterated into a cellulo-fibrous, ligamentous string, which in the course of time disappears still more, until it can no longer be recognised. This alteration is effected in smaller vessels in from about twenty to twenty-two days, and in larger ones in from thirty to forty-five days.

The ligatures by which the middle circular fibrous coat and the lining membrane have been originally divided, are loosened, and come away, in consequence of the suppuration of the cellular sheath at the spot where they have been applied, during the above mentioned process.—This suppurative process not unfrequently gives rise to destruction of the coats of the vessel above the ligature, and of the thrombus, and hence induces hæmorrhage.

The circulation is established, after the application of the ligature, in the same manner as in obliteration of an artery generally, by the dilatation of the lateral branches and their anastomoses,—the so-called collateral circulation, which is developed, in cases of spontaneous and gradual obliterations, with a rapidity proportional to the increasing contraction of the vessel, so that its final occlusion is imperceptibly effected.

When a main artery has been tied, the circulation is at first carried on by means of all the innumerable communications of the small ramifications; subsequently, however, some of these vessels and anastomoses dilate in a preponderating manner, while the others gradually return to their normal

calibre. This dilatation is especially remarkable in the small branches, whilst the trunks and larger branches are relatively dilated to a very inconsiderable degree. (Hodgson.)

We would only add to this description the materials derived from a review of the facts and opinions which have been deduced from an investigation of tied arteries in man, and from the simultaneous consideration of highly important conditions analogous to thrombus.

We must, however, at once premise, that we do not regard this question as wholly settled, since this process in man presents numerous anomalies, independently of those cases in which the thrombus is not duly formed, in consequence of debility, cachexia, &c.

Our views in reference to the process, and the individual conditions on which it depends, are as follows:

1. We are of opinion, *that the occlusion of a tied vessel may take place without the occurrence of thrombus (the inner plug),* and that this is a mere incidental formation, and not by any means an inevitable and necessary condition of obliteration. There is very frequently no thrombus present, and occasionally its place is supplied by an adhering red gelatinous, in general irregularly thick, wrinkled, gland-like, shaggy coagulum, whose colour is subsequently changed to a yellowish-red tinge; or the thrombus is inadequate to fill up the whole of the vessel, and hangs loosely in the stump of the artery, without actually adhering at any one point; although, notwithstanding this arrest of growth in the thrombus, it yet closes the extremity and a neighbouring portion of the vessel. No trace of the previous existence of a thrombus can, however, be detected on cutting through this coalescence.

2. Although we would not wholly deny the point in reference to every case, we are yet of opinion *that a true arteritis, with exudation on the inner surface of the vessel—constituting the so-called adhesive exudation, by which the thrombus is fixed and made to adhere to the wall of the vessel—is not an essentially necessary condition;* since we have found it absent in numerous cases, in which all the requirements for occlusion were present,—that is to say, where neither a change, induced by exudation in the tissue of the circular fibrous coat and in the lining membrane of the vessel, nor a free exudation on the

latter, could be distinctly recognised. We do not regard the albuminous or gelatinous layer, which invests the thrombus and the lining membrane of the vessel, and attaches the thrombus to the arterial wall, and which at first is transparent, but subsequently becomes white and opaque, as the product of arteritis, or as an exudation, but as a product of the blood—as a structure analogous to the lining membrane, (See p. 270,) which is produced in the stump of the vessel with a readiness proportional to the necessary mechanical conditions which are present. We may very often distinctly perceive how it encloses the thrombus, and extends, sometimes in a bridge-like form, from its basis towards the wall of the vessel. In other more advanced cases, these two lamellæ are everywhere, or at some spots, fused together; and, in the latter case, as the thrombus does not completely fill the vessel, it adheres by thread-like structures or bridges. The wrinkled, velvety coagulum investing the inner wall of the vessel, and which we have already described, has a similar significance; in like manner we believe, that the degeneration which attacks the circular fibrous coat, and gives rise to loosening, bleaching, and lacerability, is not to be regarded as the consequence of an exudation, and to be referred to its action on the tissue, but must be considered in the light of a regressive metamorphosis—an involution,—such as we meet with in atrophies of arteries, as, for instance, the obliteration of the foetal passages, &c., which are effected without the agency of any inflammatory process.

3. We hold that *the occlusion and obliteration of tied arteries are essentially dependent on the same process that occurs in vessels which no longer receive an energetic current of blood, in consequence of the circulating fluid taking another course, and become unserviceable*, as, for instance, the umbilical arteries and the Ductus arteriosus. After the end of the tied artery next the ligature has become closed by the fusion of the opposite surfaces of the inner wall of the vessel, the further obliteration follows from the decrease of the vessel as the blood is turned into another course, and a collateral circulation established; and from its walls finally coalescing, either by means of the original lining membrane, or of a newly deposited layer of that structure. The white mass, which we find as a central sub-

stance in the stump of the vessel, seems therefore to consist of this newly deposited stratum.

When a thrombus is formed, which is far more commonly the case, the same process takes place—that is to say, the vessel contracts, and becomes occluded above it; it undergoes a metamorphosis into a fibroid string—a white fibrous mass.

4. *Whether the thrombus, in certain cases, disappears by re-sorption into the mass of the blood in a state of minute disintegration or (as Remak expresses it) by solution, is a point which is certainly not at present established.* There are, however, no facts positively opposed to this view, and it would be an occurrence in whose favour there are many analogies,—as, for instance, the fusion or resolution of coagula of blood in inflamed veins, the diminution and the final disappearance of vegetations on the valves of the heart, the disappearance of the ends of phlebolites, &c.

5. We have never observed the formation of vessels in a thrombus (its so-called vascularisation). We do not, however, in the least doubt the accuracy of Stilling's observations,—that is to say, that the mass of the thrombus was porous, and capable of being injected; we cannot, however, participate in his view, that this condition depends on a true formation of vessels, and represents an organisation of the thrombus. We prefer believing that *this condition is the same as that with which we have become acquainted as channelling of the deposit, (of the structure analogous to the lining membrane of the vessel, and formed in great excess, See p. 264,) and which we regard as a very remarkable phenomena; that even this channelling sometimes occurs in other structures similar to thrombus, as, for instance, in the fibrinous coagula in the heart; and that it is of this, and nothing else, which observers speak, when they fancy they have injected polypi of the heart. (Alex. Thomson, Vernois, See p. 215.)* We have recently had opportunities of observing this porosity, and the cavernous structure to which it gives rise, in vegetations within the cavities of the heart.

Holding this view of the case, we cannot regard the diminution and shrivelling of the thrombus, whereby its vessels—that is to say, these canals—become obliterated, as a regressive formation in Stilling's sense.

6. Neither have we had an opportunity of observing *a long central blood-vessel, either single or ramifying at its extremity, running through the stump of the obliterated vessel*, as described by Lobstein and Blandin; nor have we ever observed the arborescent sprouting of vessels from a stump, as seen by Jones, Ebel, and others. According to our view, this phenomenon is intimately connected with the channelling of the thrombus, and the presence of these central vessels in the stump is exclusively owing to the persistence of longitudinal canals in the thrombus, such as have been frequently noticed by Stilling; and these arborescent vessels are nothing more than such persistent canals of the thrombus, which may perhaps, in the course of time, become longer and broader within the atrophying stump of the vessel. They most assuredly have no affinity with true vessels, however generally they may be regarded in that light.

We think it highly probable that Mayer's case of two arch-like lateral vessels, which connected the two extremities of the carotids after they had been tied, belongs to this class, although we are unable to give a definite opinion on the subject.

The formation of central canals in the coagula which obstruct inflamed veins—under which head we must include the case observed by Barth, of a central canal through an old plug obstructing the abdominal aorta,—may depend upon the same process of channelling, or upon another process, to which we shall refer, when we proceed to the consideration of the veins.

IV.—ABNORMAL CONDITIONS OF THE VEINS.

§ 1. *Deficiency and Excess of Formation.*

We have already noticed, under the head of Anomalies of the Heart, the most important anomalies and other deficiencies of structure affecting the trunks of the venous system. Moreover our remarks, in the corresponding chapter on the Arteries, apply likewise to the Veins.

§ 2. *Anomalies in their Origin and Course.*

Various anomalies of this nature are of frequent occurrence in the venous system, although they do not, according to

Meckel, preponderate over those of the arteries to so extensive a degree as is generally supposed. We refer our readers to the more circumstantial anatomical works for a detailed notice of these anomalies.

There is, however, one form of anomaly belonging to this class which deserves especial mention, notwithstanding the notice which will be given of it under Dilatations of the Veins. This form consists in an anastomosis of the epigastric cutaneous veins with the umbilical vein at the navel, on which depend the persistence and patency of the latter vessel.

§ 3. *Diseases of Texture.*

We purpose, for the better comprehension of the subject, prefacing our consideration of other anomalies, as, for instance, those of calibre, by a notice of these diseases.

a. Inflammation.—The study of inflammation of the veins (*Phlebitis*) constitutes one of the most important departments of pathology. It is entirely the result of anatomical research; yet, however complete may appear to be the development which this subject has attained in our day, it still presents many important deficiencies, which have either been disregarded by observers designedly or from a deficiency of materials, or have been supplied by irrational conclusions and hypotheses.

Inflammation of the veins is a very frequent disease, and is highly important, both on its own account, and also more especially from the absorption of its products into the blood. It is, under all circumstances, incomparably more frequent than inflammation of the arteries.

Its seat is the cellular coat of the vein, and likewise the circular fibrous coat, in as far as the latter exhibits a certain degree of vascularity; and its products are deposited alike in the tissue of both these coats, and in the non-vascular strata of the lining membrane of the vessel, from whence they extend to the canal of the vein.

It more frequently exhibits an *acute* than a chronic character, and it is then distinguished by the deposition of exudation on the inner surface of the vessel. The following remarks refer to this form of the disease, the *chronic* form of which will be considered in a future page.

It is especially necessary to distinguish *two forms* of phlebitis:

1. *Phlebitis* (*inflammation of the coats of the veins*) is the *primary disease*, although it may be owing to various causes, while every anomaly of the blood within the inflamed tube of the vessel, and still more, perhaps, beyond that spot, such, for instance, as the coagulation of the blood within the inflamed vessel, is a *secondary phenomenon*, depending upon the product of the inflammation.

This phlebitis is very frequent as a *primary disease*, and arises from the most various injuries, as cuts or thrusts, affecting either the vein alone, or, conjointly with it, other soft and firm parts; from contusions and displacements of different soft parts including the vein; or from many forms of surgical or medical maltreatment of wounds of the veins. This disease so far depends upon the epidemic constitution, that it is of extremely frequent occurrence at certain periods, with or without the concurrence of these favouring circumstances. This disease may also be of a *secondary* character, and in that case it is derived either from inflamed contiguous structures—as, for instance, the inflammation of the veins in the neighbourhood of abscesses, phlebitis from inflamed carious bones, &c.,—or is of a metastatic nature, as the phlebitis which occurs in the course of many different acute febrile affections, and as one of their sequelæ.

2. *At other times the coagulation of the diseased mass of the blood within the tube of some one vein is the primary, and indeed the special occurrence, which gives rise, from reaction as it were, to inflammation of the coats of the veins—phlebitis.* This disease is then a *dependent, secondary affection, of subordinate importance.* Such a form of phlebitis always consists in the establishment of a disease of the blood, which is either of a spontaneous character, or depending upon the absorption of different deleterious substances, such as inflammatory products originating either within or external to the vascular system. It constitutes the most frequent of what are termed metastases, especially if we include the process of the coagulation of blood in the capillaries—the so-called *capillary phlebitis*.

This distinction of character, which has hitherto not been

sufficiently regarded or properly understood, is of the greatest practical importance and interest, since it affords a clue to the right comprehension of the significance of phlebitis in individual cases, and thus contributes to throw light on many points in the history of phlebitis which had either remained entirely obscure, or had been explained in a wholly irrational manner. We shall always indicate this latter form of phlebitis as that which depends *upon coagulation of the blood*.

A. The following are the anatomical indications of (acute) phlebitis:

1. *Injection and Redness of the Cellular Coat of the Vein*, in different degrees and shades of colour. The cellular coat is very commonly intersected by varicose vessels, and is at the same time of a bluish red colour, which, however, experiences various modifications by the infiltration of inflammatory products into its tissue. The latter membrane very frequently presents a darkish red, mottled or streaked appearance, in consequence of slight extravasations.

2. *Infiltration of the Cellular Coat*, with a serous, sero-fibrinous, partially solidifying, greyish, grey or yellowish-red moisture, and bulging of the coat; the infiltration is very commonly associated with a thin sero-purulent, or thick purulent moisture and with bulging, whilst more or less circumscribed abscesses occur in the interior of the vessel.

The neighbouring cellular tissue participates, in various degrees, in the process, although, generally speaking, in proportion to its vicinity to the seat of the disease; the tissue becomes vascularised, infiltrated, and swollen, and the vein becomes then fixed or imbedded in it. We also very often observe circumscribed abscesses, together with diffused purulent infiltration at some distance from the vein.

3. *Injection and Redness*, as well as *the other discolorations* produced by the infiltration of various products, *extend into the circular fibrous coat of the vein*. The latter coat appears to be vascularised; but more frequently the injection and redness of the tissue are already obliterated. It then presents a greyish yellow faded appearance, and is discoloured at different points by imbibition from within, or from the contiguous extravasations in the cellular coat, or is mottled red by small extravasations within its own tissue; it is, moreover, unusually

succulent, and is swollen. In phlebitis with purulent exudation, it is most distinctly infiltrated with the purulent or sero-purulent fluid.

4. *The inner coat of the vessel presents a dirty-white appearance, or is coloured red, violet, brown, or even green, by the imbibition of hæmatin from its interior, or from hæmorrhagic exudations into its tissue. It is swollen; the inner surface is devoid of lustre, is dull, felt-like, and wrinkled. When purulent exudation is present, it is more especially of a pale-yellowish colour, succulent and lustreless.*

5. In addition to these alterations, *all the coats of the vein are relaxed in their texture, and admit very readily of being torn, and separated from one another.* In some cases the strata composing the inner coat (together with the valves) are detached from the circular fibrous coat, and even cast off in the form of a tube; and this is of very general occurrence in the more intense forms of phlebitis with purulent exudation. —In these cases, the inner coat, which is thrown off in the form of a tube, may be the more readily mistaken on a superficial examination for a tubular exudation, when its tissue admits of being readily torn, and it has been coloured yellow by the imbibition of pus.

6. *The vein appears to be dilated and paralysed; its tube is generally either filled by a plug of blood, which either resembles a recent coagulum, or has entered into different metamorphoses, or is filled with the product of the process (the exudation), more especially pus.* The formation and significance of the above named coagulum are intimately connected with the actual process of exudation, as will be seen by the following remarks:

7. *Exudation.*—The exudations deposited in the texture of the venous coats, and in the contiguous tissues surrounding the vein (its cellular bed), have already been in part considered, both in reference to their bulk and nature. A far more important class of exudations are *those which are deposited on the inner free surface of the vein (within the vein), and which, owing to their absorption into the blood, and the infection to which they may give rise, impart to phlebitis the dangerous character that renders it so formidable a disease.* The general disease arising from these conditions, together with its intensity

and character, depends upon the nature of the exudation, and also upon certain accidental circumstances, which we now proceed to consider.

a. The exudation may vary very considerably, both in reference to its physical properties and its internal composition. A direct anatomical demonstration of the exudation itself, either in reference to the originally inconsiderable quantity in which it appears, to its absorption into the blood, or still more in regard to the evidence of its original and special quality, is very difficult or even impossible. We would especially notice :

The so-called plastic exudation, capable of undergoing a metamorphosis of tissue, which, in rare cases, occurs in an appreciable quantity as a flocculent, soft, or consistent membranous coagulum on the inner surface of the vessel, or adhering to the fibrinous plug which fills the vein. The quantity of exuded serum originally contained within it never admits of being detected, as it is absorbed by the blood at the moment of its exudation, together with the greater portion of its coagulable matter.

The purulent and ichorous exudation, which is a very frequent product of phlebitis, is generally secreted in such abundance, that it may be easily recognised, even in those cases in which the blood has coagulated within the vessel. It very commonly expels the blood entirely from the vein, which is then completely filled with pure pus or ichor, the product of the process of exudation. We, moreover, here meet with exudations of a fibrinous, purulent product deposited under the lining membrane of the vessel, in the form of islands or large patches, exhibiting diffused, purulent, ichorous infiltration of the venous coats, together with discoloration, loosening, and a high degree of lacerability and detachment of the strata composing the lining membrane, in the form of a lax, lacerable, disintegrating fusing cylinder, which might easily be mistaken for a tubular exudation.

Are any of these exudations of a hæmorrhagic or a tuberculous character? In many instances, indeed, we observe a red, brownish or violet red, or chocolate brown coloration of the exudation on the inner surface of the vessel, together with a hæmorrhagic suffusion of the coats of the vein, and centres of hæmorrhagic exudations in the neighbourhood of the vessel.

We have never observed a tuberculising exudation, or even one whose nature led us to suspect a tuberculous character, on the inner surface of the vein. (See our subsequent remarks on Tuberculosis.)

δ. The coagulation of the blood in the inflamed vessel, or the formation of a fibrinous plug, which is most intimately connected with the deposition of an exudation upon the inner surface of the vein, and with its absorption into the blood, is a phenomenon of the greatest and most varied interest. It arises from the contact of the blood with the products of inflammation. The subject has been already generally considered under Diseases of the Blood, and will therefore be noticed here only in as far as is indispensably necessary towards the right comprehension of phlebitis.

Phlebitis, if we may judge from appearances after death, very rarely occurs without a simultaneous coagulation of the blood in the inflamed vein.

The inflamed vein is very commonly filled by a cylindrical fibrinous plug, which, according to circumstances, is either single or ramified, and terminates conically at both extremities.

In phlebitis having a purulent exudation, the coagulum is either present in the above described form, or is disintegrated, and blended with the purulent product in the form of loose, friable detritus; or, finally, there may be no trace of its presence, in which case the vein is entirely filled by copiously exuded pure pus.

8. Further evidence of the phlebitic process, both in reference to its own nature and that of its products, and to its highly important character, is afforded by numerous secondary conditions, which we shall briefly notice in the following remarks, referring our readers to our previous observations on the subject in Diseases of the Blood.

a. The immediate consequence of the phlebitic process is a diseased condition of the blood, arising from the absorption of the morbid products, which constitutes the basis of all the subsequent secondary phenomena. This disease generally induces degeneration of the blood, according to the character of the product, either into a so-called phlogistic condition (hæmitis, hyperinosis), or into pyæmia. Hence arise:

b. The processes of stasis and coagulation of the blood in

various portions of the capillary system (lobular processes, deposits, metastases, capillary phlebitis), with the different metamorphoses of such a coagulation; namely, shrivelling to a fibroid callus with atrophy of the tissue, or purulent, ichorous fusion with similar destruction, necrosis of the tissue; *processes of coagulation in larger vessels, more especially the veins*; and finally in the *heart itself*, under the form of different vegetations.

c. The allied processes of stasis and of exudation into the parenchymatous structures, as well as upon the membranous, serous, and mucous surfaces, with the fusion of the substratum, which is especially perceptible on the mucous membranes, and with suppuration and necrosis of the tissue.

This general infection of the blood by the product of phlebitis, together with the further phenomena depending upon that process, does not, however, invariably take place,—a circumstance that some observers attempt to explain, in imitation of Cruveilhier, by the so-called *sequestration of the vein*. Thus, for instance, the coagulation of the blood on the limits of the inflammation, and the exudation into the vein, are supposed to isolate the inflammatory product—the pus—and prevent its absorption into the blood.

We have been led, by extensive experience, to adopt the following views in reference to the solution of these two intimately associated questions regarding the cause of the non-occurrence of a general infection, and the significance of the so-called sequestration, as a special means of arresting the process.

The non-occurrence of a general infection of the blood in phlebitis would appear to be frequent, if we judge from observations on the living subject; but, on the other hand, it is rarely noticed after death, where the phlebitis which is brought under our observation is generally characterised by purulent exudation.

In the latter cases, therefore, the exudation must be absorbed into the blood, and carried away with it from the seat of its formation.

The reason of the non-occurrence of a general infection depends, in our opinion, upon the fact of the blood coagulating at the place of the exudation, and upon the rapidity with which

the whole of the blood, or one of its strata, is coagulated in consequence of the absorption of the inflammatory product; whence the course of the recently deposited or still exuding product is at once arrested.

As, however, this coagulation in the ordinary and more frequent cases is not effected immediately, but requires (as we see exemplified in the frequent development of coagula in a section of the vascular system remote from the infected portion of the blood) that the heterogeneous substance must remain for some time in contact with the blood, we are able to explain why a portion of the exudation is, in general, carried onwards by the circulation, and the blood is then infected before the coagulation can be established in the vessel.

A sequestrating fibrinous plug must be distinguished from the *coagulum originally filling the vessel, and induced in the blood-current by the absorption of the exudation.*

It is certainly true, that in every case of phlebitis the coagulation of the blood extends beyond the limits of the inflammatory centre, along the vessel, and the coagulum filling it. In order to comprehend the significance of this coagulation as a means of sequestration, it will be necessary for us to form a clear idea of the conditions requisite for its formation.

The coagulation is effected in a simple manner, above and below the inflamed vein, and around the coagulum which originally filled it. The blood is coagulated below the inflamed vein (at its circumference) in all the branches where it is retained by the coagulum obstructing the vessel; above this point, towards the centre, the blood is arrested as far as the next considerable-sized venous branch that opens into the diseased vein. The coagulation is thus dependent on the coagulum originally obstructing the inflamed vein, and is essentially a thrombus.

Seeing that this form of coagulation can only be effected after the formation of the original coagulum, there are two points to be considered in reference to its significance.

a. A general infection usually occurs, as has been already stated, before the development of the coagulum which originates in the absorption of the exudation, and consequently still longer before the formation of the sequestrating clot.

b. The original coagulum is rapidly formed after the deposition of the exudation, and it hinders the general infection by entirely filling up the tube of the vessel, and absorbing the whole of the exudation. The sequestering plug does not appear, in either case, to be of any essential service.

We here, however, draw the following inferences in reference to the possibility of infection arising from the metamorphosis of the original coagulum :

1. If the phlebitis had deposited a so-called plastic exudation, and belonged to the form which terminates in disintegration or obliteration (See the modes of termination of Phlebitis), the sequestering plug would be of no obvious utility in either of the cases considered under *a* and *b*.

2. If the phlebitis had deposited a purulent ichorous exudation, there would necessarily have existed one or other of the following conditions :

- a.* A general infection of the blood must have been induced previously to the development of the original coagulum, which may either have filled the diseased vessel throughout its entire length, or may have been limited to the margins of an accumulation of pus in the vein, if that fluid were exuded in large quantity. The sequestering plug cannot, in such a case, hinder the pyæmia, in the course of whose existence it has, in fact, been developed ; while it is itself, moreover, subject to purulent fusion from a prolonged continuance of the disease.

- b.* Or the original coagulum may have been rapidly formed by coming in contact with the pus, in which case general infection of the blood could not possibly have been effected at that moment, but inasmuch as this coagulum undergoes a more or less rapid purulent fusion, there is a possibility of the blood becoming secondarily infected by the disintegrated admixture of the coagulum. This coagulum may be permanently retained through a subsequent coagulation of the blood that has continued unaffected. It is only under these conditions that the coagulation which occurs at the limits of the inflamed vein is of essential use—that is to say, *it is only the pus proceeding from the metamorphosis of a coagulum established in the vein by the absorption of a purulent exudation produced at the spot, that can in the true sense of the word be sequestered.*

In addition to the signs and consequences of phlebitis to which we have already referred, there are certain *associated and consecutive phenomena*, which still require notice; namely, accumulation of blood in the small veins and capillaries beneath the inflamed vein, a cyanotic tint, and œdema around this portion of the vascular system; these are consequences of the occlusion of the inflamed vein. As the inflammatory process extends, we have inflammation of the cellular sheath of the vein and of the surrounding cellular tissue, with sero-fibrinous, sero-purulent, hæmorrhagic exudations; we further have inflammation of the skin in the form of erysipelatous redness, which, from the beginning, accompanies the inflammation of the subcutaneous veins in the form of red streaks along the course of these veins; and, as a final result, we have moist gangrene, caused by the stasis established in the capillaries by the extensive occlusion of the inflamed vein.

The following are the *terminations of phlebitis*: it may end in resolution; in chronic inflammation, with persistent thickening; in coalescence of the vein with contiguous structures; and in dilatation, in persistent obliteration, or in suppuration of the vein.

1. The *termination in resolution* (or perfect recovery) is by no means rare in slight cases of phlebitis, even when there has been a general infection,—like that occurring in endocarditis, which, independently of the local residua, very frequently assumes a similar favorable character, notwithstanding the pre-existence of general infection. The coagulum obstructing the inflamed vein is gradually absorbed into the blood in a finely comminuted state, and being dissolved like the originally absorbed exuded substance, the diseased vein becomes again free.

2. In some cases there remains a *condition of chronic vascularity of the coats of the vein, with bulging*, which is not unfrequently accompanied with a rusty-brown or slate-grey discoloration, with paralysis and dilatation of the vein. In this condition there are often acute relapses, more especially in the veins of the lower extremities. It finally leads to hypertrophy of the coats of the vein, in consequence of the continued accumulation of the exudation in their tissue; to rigidity of these coats; to permanent dilatation, and, by means of the

sclerosis of the surrounding cellular tissue, to permanent immobility of the vein, and coalescence with the neighbouring structures—as, for instance, aponeuroses, muscular sheaths, the general investments, the periosteum, &c. The latter condition further predisposes to the formation of new layers of lining membrane from the blood, in consequence of the retarded flow of the blood-current.

3. *Termination in obliteration* is induced by means of the coagulum, which obstructs the inflamed vein. This coagulum, which is formed by the absorption of a so-called plastic exudation (that is so say, an exudation capable of being metamorphosed into tissue), undergoes a gradual decoloration and is converted into a whitish, fibroid band, which is very commonly interspersed with rusty-brown or black pigment. After this metamorphosis, the coagulum becomes shrivelled.

This string or band is attached to the wall of the vein, either at all parts of the circumference, or only at separate points, by means of a cellular structure, formed by the metamorphosis of a portion of the exudation on the inner surface of the vein. If the wall of the vein participate, in the former case, in the shrivelling of the coagulum, the vessel is rendered impermeable, and becomes finally atrophied, and *completely obliterated*. But if, on the other hand, the wall of the vein does not generally participate in the shrivelling of the coagulum, in consequence of the latter being attached merely by partial adhesions, or if the blood forces its way into the obstructed vein, notwithstanding the total adhesion of the coagulum, the structure by which the shrivelling coagulum is attached to the wall of the vein becomes torn into threads and laminae, which gradually acquire an investment of recently formed lining membrane from the blood; and the vein being in consequence only partially obliterated, exhibits the following appearances:

The vein is either occupied by a fibroid, roundish string, which, adhering only to a portion of the wall of the vessel, leaves the latter free and permeable in other parts.

Or, in addition to this connection of the fibroid string with the vessel, thread-like bridges, or membranous partitions, which are more or less perforated—the torn adhesions above referred to—are also attached to the free portion of the wall of the

vein, while its tube is broken up into numerous straight or oblique canals or divisions.

Or the fibroid string is attached at various parts to the interior of the vessel, by means of adhesions, arranged in the most irregular manner.

The coats of the vein are thickened, the vessel itself being more or less firmly imbedded in a cellular stratum in a state of sclerosis; the free portion of the wall is dilated and elongated, so that the vessel describes intestine-like coils round the resistant fibroid string, or twists itself around it as around an axis, as the former changes the points of its adhesion. This condition gives rise to a special form of varicosity. The vessel at the same time presents some analogy in its calibre with the structure of the sinuses of the dura mater, more especially with the superior longitudinal sinus.

This fibroid string within the vein may ossify in the progress of time, constituting a form of central ossification.

This condition has been more especially observed in the cutaneous veins of the lower extremities—on the trunk and ramifications of the saphena veins. We noticed it in one case, together with the products of intense peritoneal inflammation, on the whole system of the mesenteric veins; and on account of its rarity, we will give a brief history of it.

The body of a girl, aged 13 years, exhibited the following appearances, in addition to an excessive degree of emaciation, and a pale decoloration of the general investments. The abdomen was swollen, and felt hard and board-like to the touch; the linea alba presented the resistance and the appearance of a cartilaginous layer, and was a line and a half in thickness; the same character was generally exhibited by the aponeurotic portions of the abdominal wall. The peritoneum of the abdominal wall was invested by a pale slate-grey, cartilaginously-tough pseudo-membrane, a quarter of a line in thickness, which extended to the intestinal canal and the stomach, which it covered. It contained a yellowish-white purulent fluid. The mesenteric vein, with its ramifications, was partly thickened and callous, while its canal was divided by numerous bridge-like partitions, which were perforated at different spots, and whose margins were torn, and was partly contracted by detached structures of this kind. The walls of the portal vein

were uniformly thickened. The coats of the hepatic, cystic, and common bile ducts were swollen, whilst their mucous membrane was covered with numerous, and generally suppurating, villous growths; the ducts contained an ichorous, brownish fluid. A similarly coloured dark fluid was also found in the stomach and the intestinal canal. The patient, in addition to general indisposition, had suffered from chocolate-coloured discharges, both by stool and vomiting, and had also, throughout her illness, vomited light-red, pure blood, which proceeded from the gall-ducts.

Cheesy matter, and a substance resembling moist chalk or mortar, are, moreover, occasionally found in veins that have been obliterated, and are either the disintegrated remains of the coagulum which had obstructed the vessel, or inspissated pus.

4. *The termination in suppuration*, and in an acute purulent fusion and necrosis of the coats of the veins, corresponding to the previous (acute) course of the disease, is, on the whole, somewhat rare. The dilated vein is filled with a large quantity of purulent exudation, mixed with blood, and all its coats, besides presenting a dirty-red tint from imbibition, are infiltrated with pus, and tear with great facility and almost like tinder; while the inner coats peel off from the cellular sheath in the form of a crumbling pipe or tube, and the cellular tissue surrounding the vein is in a state of suppuration. This process sometimes occurs at individual spots, so that, after the solution of the venous wall, circumscribed abscesses are occasioned in the adjacent cellular tissue. This acute suppuration of a vein usually only occurs when the highest degree of pyæmia has been developed. A more frequent occurrence is a protracted suppuration in sequestered varicose veins, which had originally suffered from chronic inflammation, a subject to which we shall again recur. The phlebitis induced by the coagulation of blood often terminates in *gangrenous suppuration and fusion*.

From what has been already stated, both in reference to the exudation and the last two modes of termination, it follows that the phlebitis of which we have already treated is sometimes *adhesive* and sometimes *suppurative*.

B. *The phlebitis depending on coagulation of the blood* differs from the form of acute phlebitis hitherto treated of, inasmuch as the coagulation within the vessel is the primary phenomenon,

whilst the inflammation of the coats of the veins—phlebitis—is associated with it merely as a secondary affection. The coagulation is therefore not occasioned by the inflammatory product of the coats of the veins—that is to say, by the absorption of the exudation into the blood from the inner surface of the vein, but is the result of a disease of the blood, which is either spontaneous or occasioned by the absorption of different products of stasis or inflammation, deposited either within or external to the vascular system. This disease reaches so high a degree of development at different points, and in the second case at different distances from the centres of infection, that the column of the blood coagulates more or less rapidly, with a more or less complete separation of the fibrin. When the coagulum is once formed, inflammation of the coats of the veins, if not invariably and very rapidly developed, is at all events of very common occurrence.

The existence of this process, when developed in the manner above described, proves the undoubted occurrence of coagulation of the blood in the various portions of the vascular system, from the centre to the capillaries, even where there is no trace of inflammation in the vessel; but it does not prove that where inflammation of the vein is present, its intensity and development have been sufficient to cause the coagulation by the deposition of exudation on the inner surface of the vessel.

The indications of this phlebitis are, in general, identical with those observed in the inflammation of the veins which we have already considered, but it is nevertheless of the greatest importance to notice the following special points:

1. We very frequently observe the above indicated want of relation between the nature and metamorphosis of the coagulation and the degree of intensity we should expect to meet with at the beginning of true inflammation of the coats of the veins. The disease generally, however, exhibits a very slight intensity, while the lining membrane of the vessel is not in a condition which would seem to indicate the immediate pre-occurrence of an exudation into its-tissue, extending by means of the latter to the inner surface of the vessel. This is the more remarkable, since, as we shall see by the following facts, it is usually owing to a purulent exudation that the process is developed in its subsequent course.

2. In general it is an ordinary pyæmia which occasions the coagulation of the blood in vessels of considerable calibre. In accordance with this view, the coagulum commonly undergoes a purulent metamorphosis, a fusion into a more or less organised pus or ichor. According to the circumstances of the case, the vein finally contains a chocolate-brown, greyish red, or yeast-yellow purulent fluid, mixed with partially dissolved fragments of the plug; or a dirty-brown, brownish green, fœtid ichorous fluid, or even a very discoloured, stinking, gangrenous ichor (Phlebitis septica). The contents are here (See p. 399) not the product of the inflamed venous wall, but proceed from the metamorphosis of the coagulum.

3. In consequence of this metamorphosis—that is to say, of the contact of the inner coat of the vessel with the deleterious substance and its subsequent imbibition, the inflammation of the coats of the veins rapidly attains a high degree of development, and gives rise to corresponding purulent and ichorous exudations, which are added to the above described contents of the vein.

4. The diseased condition of the blood is in a high degree the controlling cause, while the so-called metastatic processes, to which it gives rise, are distinguished by their number and intensity.

5. An isolating or sequestering coagulation cannot, as is obvious, be in any way conducive to this process.

It must be remarked, in reference to *the terminations of this phlebitis*, that—

1. The ordinary termination, in case death be not sooner induced by the general disease, is *an acute ulcerous fusion, a gangrenous and ichorous destruction of the vein*, arising from the process already considered under 2.

2. It is very rare, in accordance with the facts above referred to, for the disease to terminate in *permanent occlusion, or in complete or incomplete obliteration*. When the pre-existing inflammation of the coats of the veins has attained so high a degree of development as to cause a plastic exudation to be deposited on the inner surface, it will give rise to the adhesion of the obstructing plug; this may also be effected by the direct coalescence of the lining coat of the vessel with the plug.

To this form of phlebitis belong also those processes in the

capillary system of the different tissues which have been commonly designated lobular processes, metastasis, and capillary phlebitis by the French. They are, in fact, the same process which we have already considered as that form of phlebitis which is induced by coagulation of the blood. We shall revert to this subject when we enter upon the consideration of the diseases of the smaller vessels, and of the true capillaries.

Although we think we need hardly enter upon any special discussion of the differential diagnosis of these two forms of acute phlebitis, after having considered them with every possible attention under the heads A and B, we would yet draw attention to the following additional remarks:

The division of phlebitis into an *adhesive* and a *suppurative* form is well known, and has been generally followed. The two forms we have established may participate in either of the above characters. We would, however, expressly notice the error into which French observers have fallen, in regarding the purulent mass, which is in the centre of the coagulum, as the product of the inflamed coats of the veins. Cruveilhier attempts to show that the pus reaches the coagulum from without, by the capillary action of the coagulum; but this very unnatural hypothesis is quite inadequate to the solution of the question. We know, from many highly important analogies, that in ordinary cases the metamorphosis of the coagulum begins at its central nucleus, and that the purulent matter in the midst of the plug obstructing the vein cannot be regarded as the product of the inflamed coats of the vein.

c. *Chronic Phlebitis*, as already observed at p. 344, is occasionally a consequence of the acute form of the disease; it may, however, likewise occur independently of the latter.

In the latter case it consists in chronic inflammation of the cellular coat, into whose tissue, and the contiguous layers of the circular fibrous coat, it deposits its products.

The cellular substratum of the vein must obviously participate in this process. Its anatomical indications are chronic inflammation of the cellular tissue.

It gives rise to dilatation, varicosity, and thickening, through the hypertrophy and sclerosis of the cellular coat of the vein; and, secondarily, through the new layers of lining membrane

that are formed from the impeded blood-current, to the gluing of the vein into its cellular bed, to rigidity of the venous coats, and to gaping of the cut tubes; in short, it makes the veins approximate in character to arteries.

It has a great tendency, on very slight provocation, to pass into acute phlebitis.

Its most obvious causes are persistent distension and dilatation of the veins, in consequence of the impediments presented to the passage of the blood through them, besides which, it very often arises from the inflammation of the contiguous cellular tissue by an extension of the process. In accordance with what has been already stated, this condition is especially frequent in varicose veins, and in the veins of the lower extremities, where it very frequently originates in the subcutaneous tissue, which is then the seat of chronic inflammation, arising from habitual eczema.

Phlebitis very generally follows the course of the blood-current towards the heart, at all events during the early stages of its development; but yet exceptional cases are not unfrequently noticed, in which the inflammation follows a different course, and extends, during its more advanced stages, in an opposite direction.

It still remains for us to notice the phlebitis which attacks some special portions of the venous system.

1. *Inflammation of the sinuses of the dura mater* arises from injuries of the cranium, in consequence of a concussion of the dura mater near the sinus, or of a direct injury of the latter from fragments of bones, &c. It is frequently observed to arise from inflammation of the dura mater in the neighbourhood of a sinus, and from inflammation and suppuration of the bone. A tolerably frequent example of the latter mode of derivation presents itself in the inflammation of the sigmoid sinus,¹ arising from caries of the petrous portion of the temporal bone, whence it commonly spreads, with considerable rapidity, to the internal jugular vein. In some rare cases, this form of phlebitis is owing to coagulation of the blood (metastasis); and as it is almost invariably accompanied, under these conditions, by a purulent exudation, we not unfrequently find that the walls of the sinus exhibit incipient suppuration. We

[¹ The anterior portion of the lateral sinus.]

and repeatedly seen cases in which the inflammation, after attacking the cavernous sinus, had extended by means of the external maxillary to the anterior facial vein, and exhibited a diffuse swelling over the skin of the face.

In inflammation of the longitudinal sinus, the venous trunks extending into it are occluded at the convexity of the hemispheres.

2. The two cavernous sinuses occasionally become the seat of coagulation, in consequence of disease of the blood, which is either exanthematic or induced by the absorption of pus. This coagulation, it is true, gives rise to phlebitis, which, together with the inflammation of the sinus, is frequently associated with meningitis.

3. *Inflammation of the trunk and branches of the portal vein in the liver*—*Inflammation of the portal branches within the liver may be either inflammation resulting in obliteration, or may be a suppurative abscess*, as will be seen from the following remarks.

We are unfortunately led, in different spots within the liver, more or less widely diffused accumulations of a callous (cellulo-fibrous) tissue, generally in an arborescent ramifying form, and, as a result of inflammation, shows us following an obliterated branch of the portal vein. In the obliterated vessel we sometimes find a yellow cheesy matter or mortar-like inspissated plug—the remains of a fibrinous coagulum closing the inflamed vein. When these callousities are of considerable size and number, and especially when they are on or near the surface of the liver, this organ undergoes a striking change of form, and presents a lobulated appearance. As a consequence of the obliteration of a branch of the portal vein, the surrounding parenchyma becomes absorbed into these cellulo-fibrous masses, which subsequently shrivel by contracting, exert tension on the contiguous tissue, and if they reach the surface of the liver, on its peritoneal coat: so that in this case they produce cicatrix-like depressions and furrows on the surface, which give to the still unaffected part of the liver a lumpy round, lobulated appearance. This form of inflammation seems, in most cases, to be secondary, and to be phlebitis induced by the coagulation of blood.

Inflammation of the trunk of the portal vein, and of its prin-

cipal branches, with purulent infiltration, is undoubtedly of very much rarer occurrence. We have repeatedly observed it with incipient ichorous destruction, and necrosis of the coats of the vessel. It invariably gives rise to innumerable purulent and ichorous abscesses in the liver, dependent on capillary phlebitis; and also to such abscesses in the lungs, with a very highly developed pyæmia. Lambron has observed a case of pylephlebitis, which was produced by the penetration of a fish-bone through the pylorus and the pancreas into the trunk of the superior mesenteric vein. (Arch. gén., Juin 1842.)

4. *Inflammation of the uterine veins, especially after delivery*, is the most common form of this affection. It attacks the gaping venous sinuses at the points where they are torn from their insertion into the placenta, and extends from thence through the veins and their plexuses which run through the substance of the uterus towards its lateral walls. From hence it very often extends to the plexus pampiniformis and the trunk of the internal spermatic vein, and finally even to the trunk of the Vena cava; or occasionally (but much more rarely) to the hypogastric (internal iliac) vein, and from thence to the veins of one or both of the lower extremities. (Phlegmasia alba.)

It usually occurs as a *substantive inflammation of the coats* of the veins, which commences from their insertion into the placenta, constitutes an integral constituent of the exudative process on the inner surface of the uterus, and extends in the direction which we have already indicated. It usually deposits purulent, ichorous, or septic products, corresponding to the product of the uterine exudative process. It is, however, not unfrequently *induced by a coagulation of blood* at various points of the uterine veins, in the spermatic vein, the Vena cava, or in the most various and remote portions of the venous system. The exudation produced on the inner surface of the uterus, or an exudation deposited by substantive phlebitis of the uterine veins near their insertion into the placenta becomes retained in the blood, and occasions its coagulation, either at once in the immediate vicinity, or subsequently at varying distances from the centre of infection, after a more prolonged action on the blood.

The pyæmia developed after delivery is one of the most frequent and intense forms of this affection.

5. *Inflammation of the umbilical veins of newborn infants*—a phenomenon somewhat frequently combined with ulceration of the navel, and accompanied by erysipelatous redness of the skin of the abdomen, very often with jaundice, and not unfrequently with peritonitis—never, on the other hand, is followed by secondary processes of capillary phlebitis. In this respect Duplay's opinion coincides with my own. Its products, therefore, in consequence of this circumstance, do not produce any infection of the blood, which is doubtless owing to the fact, that no circulation takes place through this vein after birth.

6. *Inflammation of the Vena cava ascendens* is induced in puerperal women by the coagulation of blood, extending from the inner spermatic vein into the Vena cava. Under other conditions, it also appears to arise from a similar cause.

In addition to these special cases we must mention :

a. *Phlebitis from a wound complicated with the introduction of a deleterious substance.* The wound, besides involving other structures, affects either a large vein or only capillaries. In the former case, the phlebitis following the wound is *either* the consequence of intense inflammation and of low suppuration in the soft parts, and in the wounded vein,—the general symptoms, if they are present, being not produced by the direct absorption of poisonous matter into the wound from without, but by the products of inflammation of the injured structures, and especially of the veins: *or* the case may be altogether different; the poisonous matter may penetrate into the vein when it is opened, and be taken up into the blood,—the general symptoms and the poisoning of the mass of the blood being here developed rapidly. According to the nature of the substance, there may or may not be a coagulation in the wounded vessel; when there is a coagulation, we have a further inflammation of the venous coats.

In a wound affecting the capillaries, the poisonous substance, either in a state of purity, or mixed with the products of consecutive inflammation and low suppuration, enters the vessels either directly through their open mouths, or by imbibition through their walls; the phenomena are then either general, or, in accordance with the quality of the substance, we have a

coagulum in the vein, either near or at some distance from the wound, and inflammation extending from it.

β. *Cancerous Phlebitis* especially occurs in the uterine veins, from whence it extends into the internal spermatic, the hypogastric (internal iliac), and the femoral veins, in cases of cancer of the uterus. These veins are closed by a coagulum of cancer, especially of the encephaloid form, in various stages of metamorphosis. This coagulum finds its way into the venous blood by the absorption of the cancerous matter in various modes, either as blastema, or cancer cells, or cancerous ichor. Hence this condition, when it actually occurs as phlebitis, is that form which is induced by a coagulation of blood and its metamorphosis.

b. *Hypertrophy of the venous coats, especially of the lining membrane.*—We shall treat of this subject in the present place with the diseases of texture, as in the case of the arteries, and shall allow it to follow phlebitis, because, on the one hand, hypertrophy of the venous coats is intimately allied to inflammation of the veins, and because, on the other, without a previous explanation of the mode in which an excessive formation of the lining membrane takes place within the vein, many points in the following pages would hardly be intelligible.

Hypertrophy of the whole venous wall especially consists, on the one hand, in an augmentation of the mass, and in a simultaneous sclerosis of the cellular coat of the vein, and, on the other hand, in a thickening of the lining membrane of the vessel; an augmentation in the bulk of the circular fibrous coat is generally less marked; it is caused by the persistent impediment to the blood-current, and by the accumulation of blood in the vein, while the increased bulk of the cellular coat is principally occasioned by chronic inflammation of the vein, which is commonly perceptibly dilated, and at the same time assumes, as we have already remarked, a sort of arterial habitus.

Thickening of the inner coat of the vein is the especial result of an irregular formation of new layers from the blood; it is an endogenous production. Its occurrence in the venous system is rare, as compared with its frequency in the arteries, nor do we meet with it in the same highly developed form; moreover, in the veins it never occurs as a general constitutional disease, but is merely deposited through local conditions, such as the

impediment presented to the current of blood, and occasionally the entrance of arterial blood into the vein.

The form under which the deposition of new layers of the lining membrane appears in the vein, is principally—

a. The same as that in which it generally occurs in the arteries; it usually constitutes an opaque, white, smooth, and plane stratum, admitting of being cleft into more or less numerous lamellæ; occasionally it forms a thickish, nodular, and uneven layer; and sometimes it presents a reticulated or areolar appearance. The vein never attains that considerable degree of thickness which is so frequently observed in the arteries. In reference to its metamorphoses, we very rarely observe an ossification; in veins which lie by ossifying arteries, and are fixed in a bed of inflamed cellular tissue, as, for instance, the femoral veins, we observe the process of ossification chiefly occurring in the wall of the vein nearest to the artery.

b. As a second form of these anomalies, and one which is altogether peculiar to the veins, we must notice the *vein-stones* (*phlebolites*), which have in general not been sufficiently regarded by writers on diseases of the veins. We believe that we have recognised their true nature, and that they deserve to be considered in the present place. They are concretions of a round, oval, or cylindrical form, commonly of the size of a hemp seed, a pea, a bean, or even of a hazel nut, and of a white or whitish-yellow colour, which either lie free (as is most commonly the case) in the vein, or, if they are of more considerable size, become wedged and fixed in the vein, and entirely close it; or, finally, they adhere to, or actually coalesce with, the lining membrane of the vein, by means of a cylindrical or fusiform projecting coagulum, or through delicate membranous structures. Large phlebolites often lie in saccular pouches on the side of the vein. In some cases, this pouch, together with the phlebolite, separates from the vein, when it and the vein are closely compressed in a capsule, formed from the wall of the vessel. The vein then exhibits a more or less distinctly contracting cicatrix at the spot. When the lining and the circular fibrous coats of the capsule are gradually destroyed, the phlebolite finally lies in a capsule of cellular tissue, and this appearance may have given rise to the opinion that the phlebolite is originally developed in the cellular tissue

outside the vein. Phlebolites are of very common occurrence, either separately or in small groups, more especially in the pelvic veins; that is to say, in the vessels of the bladder, vagina, uterus, and rectum of old persons; they also occasionally appear in other portions of the venous system, and even occur in young persons. They are also sometimes met with in the spleen, and in the form of sand-like concretions in the cellular spaces of certain teleangiectases (as for instance supplementary spleens).

A careful examination of phlebolites, and an attentive consideration of the circumstances under which they are formed, yield the following results:—

On cutting through the phlebolite, we discover that it is of a concentrically stratified structure; that the innermost lamellæ are usually of a whitish-yellow colour, and the outer ones white, the former being compact and exhibiting a glass-like brittleness, whilst the latter are softer and of an earthy texture. The outermost layers are composed of soft membranes, are usually white and opaque, and exhibit at different points a gelatinous translucence. The membranous structures, by means of which phlebolites are sometimes attached to the inner wall of the vessel, are prolongations, or duplicatures of this wall, and, as it were, coatings of the peripheral layer investing the concretion. There is very commonly a roundish cavity, or, instead of it, an irregular fissure within the nucleus of the phlebolite, which is dry, and of a rusty brown, or dull yellow colour. The surface of phlebolites, in some rare cases, exhibits the appearance of being gnawed at separate points to different depths; and these spots are occasionally invested with a faded yellow, fatty, soft mass. The chemical analysis of phlebolites shows that they consist of an animal substratum, with phosphate and carbonate of lime and some magnesia.—(John, Gmelin, Lehmann, and Hasse.)

The conditions giving rise to the formation of phlebolites are, as far as we know, a retarded flow of the blood in dilated veins; thus we observe them in the pelvic veins of aged persons, and in these and other veins in young persons, in consequence of an impediment to the circulation of the blood by the pressure of fibrous tumours of the uterus, enlarged ovaries, or prolapsus of the uterus in women, or of an enlarged pro-

state gland or distended bladder in men. We have seen a case where the subcutaneous veins on the abdomen had become varicose, in consequence of anastomosing with the umbilical vein, and of its remaining patent, and were so plugged up with phlebolites, that the skin felt as if it were full of shot.

We are of opinion that there is first a slight coagulation of blood in the vein, around which there is deposited in concentric layers a structure analogous to the lining membrane of the vessel and the deposition in the arteries, and formed from the plasma of the blood. These strata generally become opaque, and ossify from the interior towards the circumference in much the same order as that in which they have been produced, or in some comparatively rare cases, they undergo the atheromatous process. The nucleus which is formed from the coagulum then shrivels, assumes a rusty brown or dull yellow colour as it dries, and leaves a cavity in the centre of the phlebolite varying according to its previous volume; or it undergoes cretification, and cannot be recognised within its calcareous capsule.

The large coagula which occur together with phlebolites in varicose veins, cannot be regarded, at least when of the ordinary size, as the first step in the formation of phlebolites. They are observed in all varicose veins, including even those in which phlebolites are of rare occurrence; nor do they always present a concentrically stratified structure. It may, however, very probably be the residua of these strata, remaining after their general solution, which furnish the nucleus for a subsequent phlebolite.

The view which we have advanced in reference to the disease of the veins considered in the present section, makes it worthy of notice, that, in addition to the differences it presents when affecting the veins instead of the arteries, this disease of the veins is characterised by the extremely rare manifestation of the atheromatous process in either of the forms of deposition from venous blood; indeed, as far as we are aware, it never occurs in a deposit of the first form, notwithstanding its great frequency in the corresponding deposition from the arterial blood.

c. Adventitious Structures.—These formations are in general alike rare in the veins and the arteries (see p. 273), although

cancerous disease of the veins presents an exception to this rule.

1. A *fibroid tissue* occurs as an inflammatory product in thickening and sclerosis of the cellular coat of the vein. It is in a great measure the cause of the resemblance to the character of an artery exhibited by the diseased veins.

2. An *anomalous osseous substance*,—the so-called *ossification of the veins*.—It may be distinguished in accordance with its seat into a *peripheral* and a *central* form. It also varies in the character of its substratum.

a. The *peripheral* includes the process of ossification that takes place in an excessive formation of the lining membrane of a vein. It appears, as in ossifications of the arteries, in the form of plates, which, however, for obvious reasons, never attain the thickness of those which occur in the arteries. Its seat is in the venous wall. It is uncommon, and, except in extremely rare cases, very limited in extent. It commonly affects varicose veins, especially the saphena, and we occasionally observe it in the deep veins of the extremities, when they are in contact with ossified arteries, as, for instance, in the internal spermatic vein in males, or in the trunk of the portal vein or its splenic branch (of which we have examples in our museum); they must be ranked amongst the very rarest phenomena.

b. *Central ossification* includes two forms of concretions, differing in their substrata, namely, phlebolites and the ossification of the coagulum closing an inflamed vein (see Phlebitis) after its temporary metamorphosis into a fibroid string. Both lie in the canal of the vessel, either freely, or coalescing in the modes that have been already described, with the wall of the vein.

3. *Tuberculosis* does not occur either in or on the blood-vessels, as we have expressly remarked in our observations on the endocardium and the arteries.

4. *Cancer* is on the other hand by no means a rare phenomenon in the veins. There are two different ways in which it may occur:—

a. The walls of a vein are very often perforated by an adjacent cancerous growth, that is to say, the cancer attacks the tissue of the vein, like other tissues, and penetrates into

the canal of the vessel. In this way, large veins, as, for instance, the trunk of the ascending Vena cava, not unfrequently become entirely closed by exuberant cancerous growths lying on the lumbar portion of the vertebral column; and the same occurs in the veins of a cancerous organ, as, for instance, the renal veins, the portal vein, the hepatic veins, &c.

b. There is another form which is rarer, but of much greater interest, which may be termed *primary cancer*, to distinguish it from the former. It occurs in a vein, generally one of the larger ones, as a broad or narrow, pedicled, roundish, pear-like, single or lobular adventitious mass, or as a cylindrical mass adhering to the whole or greater portion of the circumference, and closing the vein. It sometimes is attached only loosely to the lining membrane by means of a gelatinous blastema, or its parenchyma penetrates into the tissue of the venous wall to various depths. According to circumstances, of which the most important is the duration of the existence of the adventitious product, it presents the appearance either of a fibrinous coagulum, or of an aggregation of primary cells in various stages of development, the whole mass commonly presenting the characters of medullary cancer. This form of cancer never occurs except in association with a large exuberant cancerous growth in some other organ, or when there is a widely diffused and rapid formation of cancerous tumours in many organs, especially of intense medullary cancer (encephaloid). It scarcely ever occurs in the organism, except as a secondary formation. The lymphatics in the vicinity of the original adventitious structure are very often plugged up and closed by the medullary cancer.

This cancer of the veins doubtless occurs through the absorption of cancerous matter by the lymphatics and veins into the mass of the blood; and, independently of the latter, it may be of spontaneous origin, and may be the result and expression of the highest degree of endogenous disease of the fibrin: the circumstances under which this disease has been observed accord with both these modes of origin.

Moreover, we must here include the coagulum which closes the veins and originates from the absorption of cancerous or ichorous matter—the phlebitis arising from cancer.

§ 4. *Anomalies of Calibre.*

A. *Dilatation of the veins, Phlebectasis, or Varicosity in its wider sense*, is, as is well known, an anomaly of great importance in medicine, whether it be general, occurring as a preponderance of the venous system, or whether it be partial and local. It has attained this importance through certain views regarding its etiological relations, derived from clinical observations; and it has maintained this importance, although the more remote cause to which it has been referred,—the venosity,—notwithstanding all endeavours to elucidate it, has been as yet but little understood in reference to local venous dilatations, and to the formation of true varices. With the view of indicating the necessary points regarding the causes of phlebectasis, we shall first treat of its anatomical relations.

The Form of the Phlebectasis especially belongs to the domain of anatomico-pathological inquiry. Two principal forms may be distinguished.

1. The dilatation of the vein may be uniform and cylindrical, the distended vessel running in a straight line; its coats are either attenuated or not of a thickness corresponding to the calibre; or, on the other hand, they may be visibly hypertrophied and thickened.

2. The dilatation may be irregular, attacking the vein merely at certain spots, or at all events very much preponderating at them. It then includes *varicosity*, or *varicose dilatation of the veins*, in the stricter signification of the term.

The venous coats, if not absolutely are relatively thin in proportion to the calibre of the vessel, but after this condition has existed for some time, they very commonly thicken. Varicosity includes two varieties, which merge into one another, and are often simultaneously present in the same vessel.

a. The vein, while it becomes dilated, at the same time becomes elongated, and assumes a winding course, at first forming slightly concave arches; but gradually, as the vein expands on the convex side of the arch, the curve becomes sharper, and the dilatation exhibits itself as a sinuosity *on only one side* of the vein, while the opposite wall of the vessel is usually tense, but sometimes lying in folds. As this process may be several times repeated on the same side, or as it may alter-

nately occur on either side, the course of the vein resembles the convolutions of the intestine, or, even more closely, those of the *vesiculæ seminales*; indeed, when the vein is sharply bent upon itself, projecting ridges are produced on the interior of the sinuosities—duplicatures of the venous wall, which give to the interior a partitioned structure. We have already (in p. 346) pointed out the derivation of a remarkable degree of dilatation (varicosity) of this kind, both in reference to its winding course and the partitioned nature of its internal structure, from phlebitis, ending in imperfect obliteration.

b. When a strongly marked lateral sinuosity occurs at a circumscribed spot, a true *varix* is formed. It is a saccular expansion, which either lies with a broad base upon the vein, or is sometimes connected with the vessel by a neck or pedicle; in either case communicating with the interior by a wide or narrow opening. In relation to the construction of the walls of the varix, they are formed of all the coats of the vein; or else we observe the circular fibres separating from one another at certain spots, when the inner coat becomes so fused with the cellular coat, that if a further (secondary) bulging occur, the varix assumes a *hernial* character. The size of the varix ranges from that of a hemp-seed to that of a walnut or a hen's egg, or it may be even larger.

The varix is, as a general rule, originally roundish, but by further irregular dilatation at particular spots, it may assume an irregular, externally lobulated form; while internally it presents a cellular, many partitioned structure. Varices of smaller and more delicate veins not unfrequently present blackberry-like tumours.

The *valves* exhibit a different relation in phlebectasis. They at first offer such opposition to distension, that they limit and bind down the varix; it is not, however, by any means invariably at the valves that we find these constrictions occurring on varicose veins. The valves increase in size, to a certain degree, with the dilatation of the vein, but after a time they cease to increase, and are no longer capable of closing the enlarged vessel; they then lie, in a state of tension, transversely across the tube of the vein; or they are drawn in an eccentric direction (towards the periphery); or, finally, they may be torn, in which case they float loosely in the vessel, or they may

be almost destroyed, so that we can detect mere traces of them.

If repeated inflammatory attacks have not fixed the varicose veins in their bed of cellular tissue, and made them coalesce with the adjacent structures, they may be often readily detached, and easily raised from it, leaving furrows and cavities, with smooth, even walls.

The following are the *sequelæ and results* of dilatation of the veins, especially of varicosity.

In varicose veins, coagula of blood are occasionally observed in the form of roundish, oval, fusiform, detached plugs, or of cylinders which close the vessel. These are generally again dissolved after a shorter or longer period, and taken up into the mass of the blood; but, as new impediments, they sustain and increase the varicosity. These coagula appear to be very frequently produced in the varicose plexuses of veins, which often occur in the pelvic viscera, new clots being in the act of formation, while the older ones are undergoing solution. They lead, in the manner which has been already described, to the formation of phlebitis.

In large varices, especially such as are connected by a pedicle, we sometimes find stratified coagula of fibrin, as in aneurisms. Such varices are sometimes shut off from the calibre of the vein by a newly formed inner venous coat, investing the last formed stratum of fibrin; or they become separated from the vessel by a prolongation of their pedicle, and a closure of its calibre. In the latter case they degenerate into fibroid capsules.

Varicose veins often, however, open, either externally or into mucous canals and cavities, after they have become imbedded in the tissue of the general investments, or of a mucous membrane which has become extremely attenuated and distended over them; the vein, and the superjacent tissue with which it has coalesced, and which has simultaneously been affected with inflammation, undergoing laceration together.

The varicosity induces stases, which are either transient or persistent, according to circumstances, and are occasionally marked by exacerbations, in the capillary system; and these are followed by various forms of œdema, hypertrophy, increased

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2. Once the problem is identified, the next step is to define the objectives and goals of the project. This helps to clarify what needs to be achieved and provides a clear direction for the team.

3. The third step is to develop a plan or strategy to address the problem. This involves breaking down the problem into smaller, manageable tasks and determining the resources needed to complete them.

4. The fourth step is to implement the plan. This involves putting the strategy into action and monitoring progress regularly to ensure that the project is on track.

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... causes of variations are usually sufficiently obvious. ... the ... experimental ... the regulation of ... the ... of variations in the ... of ... the ... on a ... pressure ... a ... which may ... or even ... a ... a ... a ... of the body ... the ... of the blood into certain ... in which the ... of the veins lies in an opposite ... to the force of gravitation; excessive activity of an ... accompanied with its enlargement and hypertrophy:

adventitious products, in which vessels of considerable size have been produced—that is to say, the insertion of a new vascular apparatus into the original venous system of a part; repeated hyperæmia and inflammation of an organ; or inflammation of the vein itself. The impediments of a mechanical nature have always been regarded as of the greatest importance.

Cases are, however, occasionally met with, in which none of the above mentioned causes can be detected, and, indeed, where no mechanical hinderance can be perceived. Such cases have recently been often brought forward with the view of opposing the theory of a mechanical impediment, and of establishing other theories of varicosity. It is necessary that we should mention the grounds alleged in relation to this point.

1. The cases in which no mechanical impediment exists are by no means rare.

2. If the veins of dependent parts are more frequently varicose than others, this does not explain why they are oftener varicose in one limb than in another; or why this condition especially occurs in a particular part of a limb, and why neighbouring veins, under the same conditions, are not similarly affected.

3. Moreover, varices also occur, without any mechanical impediment, in veins in which the direction of the current coincides with that of the force of gravitation, as, for instance, in the veins of the head and neck.

4. In women, varices often occur in the legs during the early stages of pregnancy, before the uterus can exert any appreciable impediment to the circulation.

5. When constipation induces hæmorrhoids, they may be as much ascribed to the irritation and congestion which it excites as to the impediment produced to the course of the blood, for otherwise the hæmorrhoids would disappear when the bowels were freely moved, which is not the case.

6. When veins running towards a tumour become varicose, it cannot always be shown that they all undergo compression by the tumour; moreover, the blood might make its way by numerous anastomoses with deeper and non-dilated veins.

7. The blood, far from stagnating, rushes through the varices with great energy, which thus increases the difficulty of checking hæmorrhage from them.

8. The blood contained in varices is brighter than venous blood, resembling a mixture of arterial and venous blood.

9. When arterial blood makes its way into a vein, the latter becomes varicose.

10. Varices sometimes present pulsations isochronous with the heart-stroke.

11. When a main artery is tied, in cases of aneurism, we very often see varices disappear in the neighbourhood of the aneurism. (Dupuytren.)

12. Most varices extend from the venous radicles towards the trunks, which can only happen in consequence of the extraordinary impulse that the blood in those vessels receives from the heart, or of the blood flowing from the trunks towards the branches, which is always the case where a morbid formation acts as a centre of attraction for the blood.

Briquet attempted, in an unsatisfactory manner, to explain the formation of varices from an excessive fulness of the subcutaneous veins of the lower extremities, arising from the contraction of the muscles, which drive back the blood from the deep to the superficial veins; and Rima subsequently maintained that varices of the lower extremities were dependent on a retrograde motion of the blood, so that the blood flows back from the femoral into the saphena vein, and is driven from the inguinal region towards the feet by a force which is peculiar to this vein. Pigeaux, from a more general consideration of the same facts, believed that he had solved the difficulty by the assumption that varices anastomose with arteries.

If objections may be raised against some of the above mentioned facts in opposition to the theory of a mechanical impediment, we are nevertheless convinced of the untenability of the latter view. We have, therefore, after much experience, adduced other causes in explanation of dilatation of the veins. There are, however, always cases occurring in which these cannot be detected; and as varicose veins present many symptoms which are hitherto perfectly unexplained, the theory of phlebectasis is still deficient in an important part. Now, although Pigeaux's view is not yet actually proved to be correct, and is only based on the theoretical application of certain phenomena of varix, it is so far worthy of consideration, as it removes the whole question into the department of anatomy,

and thus affords facilities for renewed and more profound investigations. Thus only is it possible to obtain a well grounded view regarding the correctness of a theory which has become popular in Germany, respecting the production of a congenital (hereditary) or acquired disposition to general or local phlebectasis, respecting the production of a persistent or periodically recurring blood-crisis and its localisation, the production of peculiar critical events in the organism, in consequence of many acute processes of plethora and stasis (venous congestion) occurring from time to time in certain portions of the venous system, regarding the high importance of local phlebectases arising from them, &c.—a theory which many, even from the want (as yet) of any material information on the subject, are inclined to regard as a deception and a fiction.

Phlebectasis is most common in the prime of life. Some forms, however, develop themselves earlier, and all may persist to extreme old age. Many forms of phlebectasis may attack both sexes; some, however, chiefly, or even exclusively, affect only one sex.

Of the local phlebectases some require a special notice, in consequence of the frequency of their occurrence, and others from the annoyances and pains to which they give rise. Several very often occur in one and the same individual: whether the frequently isolated occurrence of a definite species of varicosity—as, for instance, of varicocele (Landouzy), and whether the high development of one species, in association with a low degree of another, is indicative of a vicarious action, and must be referred to a deeper cause, are points which must remain for the present undecided. As a general rule, phlebectases are almost entirely confined to the lower half of the body, and their occurrence in the upper half, must be regarded as altogether exceptional. It is almost invariably the superficial or cutaneous veins which become varicose in the lower extremities.

a. Varicosity of the veins of the lower extremities is very frequent, and may be observed in all possible stages. It attacks the system of the saphenous veins, and especially the trunk and branches of the internal saphenous vein. According to our observations, it attacks both sexes with equal frequency. A protracted, uneasy, and upright position of the body is undoubtedly a very frequent cause of this affection, although the cases are

not uncommon in which it cannot be traced to this origin. In women, especially amongst the working-classes, it is often ascribed to frequent pregnancies. Hasse remarks that, as a general rule, the dilatation commences in men in the trunk or principal branches of the internal saphena vein, while in women it usually begins in the finest cutaneous branches on the inner side of the limb. The disease is commonly first developed on the lower part of the leg, and from hence it extends to the thigh, where it is for the most part limited to the trunk of the saphena. We believe, however, that, especially in women, the cases are not very rare in which the varicosity first appears and predominates in the femoral region. It often attacks both limbs, but more commonly only one. The dilated venous reticulations either lie loosely in the panniculus, or they are imbedded in a callous, thickened, infiltrated cellular substance, with which they coalesce; while the venous coats themselves are thickened, and have a rigid appearance when cut through. This condition of the saphena vein may give occasion to the entrance of air into it, if it be opened, as is shown by an operation of Dupuytren's. Coagula of blood are very commonly formed in these veins, although it is extremely seldom that we find phlebolites in them.

Varicosity is followed by œdema, hypertrophy, repeated inflammations of the cellular tissue terminating in indurations, and inflammations of the skin, which cause it to coalesce with the subjacent cellular tissue, and give rise to an excessive formation of epidermis, and ulcerous fusion of the tissue—the so-called varicose ulcer, which is distinguished by the callosity and livor of its edges, the production of a purulo-serous corroding secretion, lax and bloody granulations, its sinuous and sometimes serrated form, and, finally, by its obstinacy. The varicose veins at its base, or in its borders, are sometimes corroded, and give rise to exhausting, or even fatal hæmorrhage.

When the pressure of the varices destroys the fascia under which they lie, they become deposited in the subcutaneous cellular and fatty tissue, and finally in the true skin itself, which now becomes extremely attenuated, inflames, and gives way, causing a hæmorrhage which also may prove fatal.

We very frequently meet with inflammations of these varicosities, which sometimes terminate in resolution, sometimes in

obliteration and atrophy—a termination which it is the object of the various operations for varices to induce,—and sometimes in purulent exudation, which may occasionally prove fatal, by giving rise to pyæmia.

b. Varicocele (Circocoele), varicosity of the veins of the spermatic cord and of the testicle is usually developed during the period of puberty, and is commonly regarded as of more frequent occurrence on the left than on the right side—a view which is borne out by our own somewhat limited experience. It begins with an uniform, cylindrical dilatation of the veins in the spermatic cord; these gradually assume a character of true varicosity, which extends downwards towards the testicle, and often affects the scrotal veins, (*circocoele*, according to Velpeau.) It is frequently combined with consecutive hydrocele, and in its higher degrees is followed by atrophy of the testicle, and at the same time by mental disease. The varicose veins are here rarely attacked by inflammation; and phlebolites are seldom produced in them, although not so seldom as in varicosities of the saphena.

c. Varicosity of the veins of the rectum, constituting Hæmorrhoids, is, next to the preceding, undoubtedly the most frequent form, and is that which has been commonly regarded as the expression or crisis of a special (often hereditary) diathesis. Although we are now fully aware that too great a latitude has been allowed to the theory of hæmorrhoids, yet hitherto no positive facts have been brought forward on which a better theory can be founded. It is worthy of remark, that even to the most recent times, erroneous views have often been promulgated regarding the nature of local hæmorrhoidal disease, in consequence of our departing from the original view, that hæmorrhoidal tumours are dilated varicose veins. We have convinced ourselves on innumerable occasions, that hæmorrhoids are ordinary varicosities; and if we must, on the one hand, reject the opinion that they are produced by extravasation and sanguineous infiltration of the cellular tissue, (Recamier, Gendrin,) we, on the other hand, find no reasons for ascribing to them a peculiar erectile nature, (Cruveilhier.)

This varicosity affects the small veins, which freely anastomose with one another at the extremity of the rectum, and which lie thickly imbedded under the mucous membrane in

the subjacent cellular tissue encircling its lower expanded portion. A certain number of these become dilated and stand out, sometimes as separate knots, sometimes as a row of nodular swellings, thus causing the lower border of the rectum to protrude; or they may be within the rectum: it is only rarely, however, that they occur above the external sphincter. In a less developed stage of the disease, and at the beginning, they are small; but, after repeated swellings, they attain the size of a bean, a hazel-nut, or a walnut. They are then protruded from the gut when the bowels are moved, the rectum is choked up with them, and they are constricted by the border of the anus, which not unfrequently gives them a pedicled form. At first the swelling disappears without leaving a trace; the oftener, however, that they swell, so much the more is the mucous membrane of the rectum, in which they lie, left in persistent folds and elongations, which project externally; and finally, when repeated inflammations have taken place, a recession of the knots (varices) no longer occurs, and they assume a condition which renders them capable of still further enlargement, but not of any diminution.

Hæmorrhoidal knots at first form roundish, simple sacs, which, however, afterwards become sinuous, and partitioned in their interior; their walls at first are thin, and collapse on being cut; they gradually, however, become thick and rigid, in consequence of repeated inflammations. One, or very commonly several fine venous branches open into the cavity of each knot, whose lining membrane is undoubtedly composed of the inner coat of the veins. The veins adhere, as a general rule, very tenaciously to the mucous membrane of the rectum, but they may be at first easily detached from the cellular tissue and the muscular coat. The cellular tissue lying between them is, like the mucous membrane, in a state of injection, reddening, and tumefaction—in short, in a condition of stasis.

The hæmorrhages which occasionally proceed from the varicose swelling of the veins in the rectum, are doubtless dependent in some cases on the rupture of a varix, and of the tense and thin mucous membrane covering it. True capillary hæmorrhages of the mucous membrane itself are, however, incomparably more frequent.

Hæmorrhoidal varices usually contain coagula of consider-

able size, which dissolve, without, however, often giving rise to the formation of phlebolites.

The stasis to which we have referred is gradually followed by a thickening and condensation of the surrounding cellular tissue, a condition which is induced in a still higher degree by repeated inflammatory attacks. It often gives rise to inflammation, whose products induce a thickening and sclerosis of the coats of the vein and of the surrounding cellular tissue, and cause their fusion with each other. This inflammation sometimes also simultaneously deposits its products within the vessel, in which case it leads to obliteration and atrophy of the varix. In rare cases, especially after the application of a ligature to the hæmorrhoidal knots, there is a purulent exudation, which extends to the larger veins, and may induce fatal pyæmia. More frequently, however, the inflammation of the surrounding cellular tissue gives rise to suppuration, abscess, destruction of the varix, ulcerous perforation of the mucous membrane, and finally to fistula in ano.

Amongst the consequences of hæmorrhoidal disease, we must also mention habitual hyperæmia of the mucous membrane of the rectum, with swelling and blennorrhœa, prolapsus ani, hypertrophy and stricture of the sphincters, sclerosis of the cellular tissue around the rectum with paralysis of the sphincters, and hæmorrhoidal ulceration of the rectum.

These ulcers at the extremity of the rectum are analogous to the varicose ulcers which occur on the feet, and often bleed very freely from corrosion of varicose veins, or even of small arteries.

Hæmorrhoids sometimes depend on certain obvious impediments in the portal system, in the ascending Vena cava, or in the heart; but in many cases no such hinderance can be detected.

We are as unable to give a strict scientific explanation of the connection between hæmorrhoidal disease generally, suppressed hæmorrhoids, &c., and the various so-called hæmorrhoidal anomalies—as, for instance, congestions, hæmorrhages, impetigo, catarrh, gout, lithiasis, &c., as we are to comprehend the true nature of what is called the hæmorrhoidal diathesis.

d. Varicosity of the vesical veins (known also as *hæmorrhoids of the bladder*) in the male sex affects the veins of the prostatic and vesical plexuses; the branches of the latter on the neck of

the bladder and around the vesiculæ seminales, external to the muscular coat, being, in general, especially dilated. In the female sex, the veins of the vagina are thus affected in addition to the vesical plexus; and the varicosity extends over the veins of the bladder to the *veins lying between the broad ligaments of the uterus*. This form of disease attains a very high degree of development, and is common in advanced life and old age. Phlebolites are nowhere so frequent, so numerous, or so large, as in the veins we have just mentioned.

The veins situated immediately beneath the mucous membrane of the bladder are less frequently varicose, although we have repeatedly observed this condition, as well as the occurrence of laceration of a submucous varix on the posterior wall of the bladder, with hæmorrhage into its cavity. The veins of the neck of the bladder present the only exception; they are frequently dilated and swollen.

Varicosity of the veins of the bladder is often combined with hæmorrhoids; in many cases, however, we find nothing but their residua, or they are even wholly absent. We are as yet unable to determine from anatomical investigations whether, in such cases, the varicosity of the veins of the bladder has actually taken the place of the hæmorrhoids, and, having thus assumed the same significance, may be regarded as of a hæmorrhoidal nature.

e. Although *varicosity of the upper extremities, and of the head and neck*, is less frequent than the above species, we yet occasionally observe this condition very highly developed in the *lips*. It is moreover of rare occurrence in the intestines, mesentery, stomach, and œsophagus. It must, however, be observed, that this remark applies only to true varicosity, since an uniform dilatation of the veins is frequently found to exist in a very highly developed condition in these structures in various affections, such as heart-diseases, impermeability of the liver, displacement of the intestines, dilatation and hypertrophy of the stomach, &c. We are, however, able, from personal experience, to confirm the observations of others, who have noticed true varices on the œsophagus, the stomach, and intestines, and even beneath the mucous membrane, which have terminated in laceration and hæmorrhage.

f. *Varicosity of the Veins of the Pia Mater* is an important

condition. It has been frequently noticed in the case of drunkards, and more especially after repeated attacks of delirium tremens, and is often combined with cerebral atrophy. The venous trunks of the infiltrated swollen pia mater on the convexity of the hemispheres which open into the superior longitudinal sinus, appear to be simply dilated below the turbid and thickened arachnoid; in the direction of their ramifications, these vessels present, however, a very peculiar appearance, being elongated, and more or less twisted into intestine-like circumvolutions, or even spirally-twisted coils. Similar dilatations not unfrequently affect the veins of the *choroid plexus*.

g. It still remains for us to notice another form of varicosity of the trunk, which affects the *subcutaneous abdominal veins*, and originates in a congenital anomaly of the vascular system. Thus, for instance, there exists in many persons an anastomosis of the umbilical vein with the veins of the abdominal integument, one of which opens into the umbilical vein. This anastomosis occasions the continued patency of the umbilical vein after birth, and thus maintains an unusual communication between the systemic veins and those of the portal system. In such cases, the veins of the abdomen become by degrees excessively varicose, undoubtedly in consequence of the circulation in the portal trunk obstructing the discharge of their blood through the umbilical vein, or perhaps even from the entrance of portal blood into the umbilical vein. This varicosity presents the appearance of a wreath-like net-work surrounding the navel (*Caput Medusæ*), or of pyramidal tumours at the side of the navel; or, lastly, the veins of the abdomen generally may be varicose in the direction of the loins and the buttocks, as well as towards the lower extremities.

Cases of this kind have been noticed by the older observers, and in modern times by Cruveilhier, Mauec, Peygot, and others. We ourselves have noticed the same condition in two men and one woman. In two of these cases the varicosity presented the *Caput Medusæ* form, and in the third (that of a man) it was present on only one side of the abdomen, from whence it extended down the loins, nates and leg to the foot. The dilated veins contained an innumerable quantity of phlebolites, varying in size from a barley-corn to a bean, which were so

densely crowded together as to make the vein feel like a skin filled with shot, (see p. 358.) It appears worthy of notice that in all the cases of the kind which we have seen, the liver was in an abnormal condition, presenting in one instance granulations, and in two others a lobulated condition, in consequence of the obliteration of several of the portal branches—adhesive phlebitis of the portal vein.

B. Occlusion, Contraction, and Obliteration of the Veins.—To this class belong several anomalies to which we have already referred in the preceding pages; and of which we now proceed to consider the most important.

1. *Contraction and final obliteration of the vein in consequence of persistent compression.*—Such a form of compression is exerted by all tumours generally, but more especially by aneurisms; and we have frequently observed both a threatened and a complete destruction of the calibre of the Vena cava superior from aneurisms of the ascending aorta. The vessel is first flattened at the spot exposed to the pressure, and when the latter is increased, the walls of the vein are at length brought into permanent contact with each other, and obliteration is thus established, consisting in a *fusion or coalescence of the lining membrane of the vessel*. The occlusion of the vessel above and below the coalescence by means of a coagulum (thrombus) is merely a secondary and unimportant occurrence, which, moreover, follows the known laws of a thrombus-formation.

2. *Occlusion of the vein from coagulation of the blood.*—To this class belong, independently of the coagula occasioned in varicose veins by the retarded flow or stagnation of the blood, the obstructing coagulum in phlebitis, and the coagulation arising at any part of the venous system from a diseased condition of the blood. Both (see pp. 345 and 350) may induce more or less complete *obliteration* of the vein. This is the most frequent form of occlusion and obliteration of the veins.

3. *Occlusion arising from phlebolites* seldom induces entire impermeability.

4. *Occlusion of the veins from cancerous secondary formations* is not of very rare occurrence, even in the venous trunks. (See p. 360.)

The ordinary results of these anomalies are *œdema and dropsies*; the latter are, however, retarded in their development and healed by the establishment of a collateral circulation (even where the main trunks are closed) through the anastomoses of numerous veins. The obstruction which the valves must here present (Stannius) is undoubtedly overcome in many cases, or so far neutralised, that a collateral circulation is established, as in the arteries, by the anastomoses of very minute vessels having no valves. The closure of the portal vein can scarcely be compensated for, notwithstanding the numerous anastomoses of its roots with the systemic veins, and hence the dropsy which it induces does not admit of cure. Closure of the Vena cava inferior is compensated for by dilatation of the Azygos and Hemiazygos, and anastomoses of the epigastric vein with the internal mammary, and of the subcutaneous abdominal veins with the axillary: it is on the other hand less easy to compensate for the closure of the Vena cava descendens, especially when the mouth of the Vena azygos has at the same time become impervious; the collateral circulation is carried on by means of the venous plexus of the spinal canal and its anastomoses with the subclavian and hypogastric veins, by means of the anastomoses of the phrenic veins with the Vena cava ascendens (and even with the great coronary vein of the heart, as occurred in a case of Reynaud's,) and lastly, by means of anastomoses of the axillary and internal mammary with the epigastric and circumflex iliac veins.

In rare cases we have also seen the circulation re-established in closed veins by means of canals, which have become developed near the centre of the occluding plug. This phenomenon either depends on the channelling of the thrombus—a process which we have already described in p. 333—or it is due to the disintegration of the central portion of the coagulum into a molecular mass, which gradually becomes taken up into the blood, while the outer layers assume a fibroid character and coalesce with the venous walls. We have never observed such cases, but they have been described by Carswell and Hasse.

§ 5. *Separation of Continuity.*—Under this head we include the various wounds and spontaneous lacerations of veins. The former, as is well known, heal by adhesion of the edges in cases of small incised or penetrating wounds; in cases, on the

other hand, where the wound is larger, or where the vein is completely cut through, the cure is effected by obliteration of the vessel, consequent on a process which is generally much the same as that which occurs after an artery has been cut through.

Spontaneous Lacerations of veins, if we except the bursting of true varices, are rare, in comparison with the lacerations of arteries. We have, however, observed them in the trunks of both Venæ cavæ, in the azygos, the pulmonary, and other large veins.

SUPPLEMENT.

Anomalies of the Small Vessels and Capillaries.—Although we deem it necessary to refer specially to the anomalies of these vessels, we must observe that we are entering upon a field of inquiry which has hitherto only been partially cultivated in reference to the general anatomy of certain processes. In the following remarks we purpose referring to what has been already stated, both generally and specially, regarding the diseases of the vessels.

We therefore omit to notice the formation of new vessels, which occurs under certain conditions, as well as the atrophy of the capillaries, which presents itself under other conditions, and proceed at once to the consideration of the following anomalies.

§ 1. *Anomalies of Calibre and especially Dilatation.*—Under this head there are commonly placed *Aneurism by Anastomosis*, *Teleangiectasis*, and *Fungus hæmatodes*.

Aneurism by Anastomosis consists, according to J. Bell, in dilatation of the small arteries and their anastomoses, giving rise to a pulsating, more or less defined, tumour; it is separated from the venous system by normal capillaries; the dilatation sometimes, however, extends to these vessels, and, finally, to the veins.

Teleangiectasis, on the other hand, consists in a dilatation of the capillaries, and appears sometimes in the form of slightly elevated patches, which may be smooth or may present inconsiderable nodules, and sometimes in the form of roundish,

nodular, and lobulated smooth tumours, which are either of a bright red colour, or of a darker, purple or cherry-red tint, and are either tough and elastic, or soft, and of a doughy consistence. They are capable of swelling and of undergoing a considerable collapse; and hence the name of *erectile tumours*, which Dupuytren has applied to them. We must, however, remark, that there is no analogy between the arrangement of their vessels and that which occurs in the erectile organs. Teleangiectasis may certainly in some cases be developed chiefly on the arterial side of the capillaries, and in other cases on the venous side; and its form may in part be referred to its position, for we regard those that extend superficially as chiefly of an arterial nature, and those which become developed into tumours as especially of a venous nature. It occurs most commonly in the tissue of the general integuments, and in the subcutaneous cellular substance; and further, besides those cases in which it is continued from these to the neighbouring mucous membranes, it is also developed independently upon the mucous membranes, as, for instance, those of the intestinal canal; and, lastly, in rare cases in other tissues, as in the muscular substance, &c.

It is in general congenital, although it is capable of further development after birth. In some rare cases it even originates in later periods of life.

The structure of congenital teleangiectasis is mainly to be referred to the transition of the dilated extremities of arteries into saccularly formed dilated venous radicles, from whence a varicose condition extends for a considerable space to the larger ramifications. The convolution of dilated vessels is held together by means of a loose and partially embryonic cellular substance. The above remarks give the main points of the opinions hitherto maintained in reference to dilatation of the minute vessels and the true capillaries—more especially when the disease is of a congenital character. But these views undoubtedly require much reconsideration. A similar observation applies equally or even more forcibly to the teleangiectases, which are observed to arise in different and more advanced periods of life, as a consequence of repeated inflammations, and of loosening and rarefaction of the tissue, in the neighbourhood of ulcers, and in and around the most various new forma-

tions, &c. The dilatation of the existing capillaries, which has commonly been considered as a sufficient cause for this anomaly, is certainly, in many cases, inadequate to explain its existence, and we find that, in addition to this condition, there occurs simultaneously a new formation of vessels,—a subject which we have already considered generally in its relation to the original vascular apparatus.

Fungus hæmatodes, which does not consist in a mere dilatation of the vessels, has been fully noticed as a secondary formation in the General Anatomy.

The present notice would be incomplete, were we to omit all reference to a morbid structure, which has been generally regarded as belonging to the teleangiectases, and from which it has been supposed, on somewhat obscure grounds, to be developed. It does not appear to us to belong to these affections, and we are rather disposed to regard it for the present, at all events, as a new formation. It includes *cavernous tumours*, having a cellular structure similar to that of the corpora cavernosa. They are composed of a cellulo-fibrous tissue and a lining membrane investing the interstices, and enclosed in a dense cellular capsule. These structures admit of being separated from the organs in which they are seated, are capable of tumefaction, and may be injected by a vein. They occur in the form of the so-called secondary spleens (which, according to Andral, are of a placenta-like texture) in the subcutaneous cellular tissue, and are of especially frequent occurrence in the liver; we have also observed them in the cranial bones, and in the pia mater. They sometimes contain sand-like concretions, which may be regarded as similar to phlebolites.

§ 2. *Separations of Continuity*.—These are very numerous in the capillaries, and arise from cuts or from lacerations induced by contusions and shocks. They result in an extravasation of blood into the tissue, and hæmorrhage externally or into different cavities and canals.

A higher degree of interest must be attached in a pathological point of view,—1, to spontaneous laceration of the capillaries in apoplexies (parenchymatous hæmorrhages) of the different organs and tissues, arising from hyperæmia (vascular apoplexy), which, although formed in the usual manner, has been deve-

loped beyond a certain limit;—and 2, to spontaneous lacerations resulting from a morbid brittleness of the walls of the vessel, or from a similar condition of boggiess in the tissue affected by the hæmorrhage, as, for instance, the substance of the uterus.

§ 3. *Anomalies of Texture.*—We have already considered the relation of the capillaries in hyperæmia, stasis, and exudation (inflammation), as well as in their modes of termination, in induration (atrophy of the tissue), in suppuration, in gangrene, and in other conditions. Although, as we must again specially remark, these vessels cannot themselves be inflamed, they yet become the seat of an inflammatory process, and a centre for the development of its products, the deleterious effects of which they are the first, under the appropriate conditions, to experience. The small veins and arteries beyond the capillaries are affected by the condition of the surrounding tissues, even where they had not themselves participated in the process of exudation: for their delicate and permeable coats are saturated and permeated by the product of the inflammatory process, on which, at least in part, the ordinary coagulation of blood within them and their occlusion depend. There then follows either a solution of the occluding coagulum and a liberation of the calibre of the vessel, when resolution of the inflammation occurs in a comparatively large vessel, or persistent obliteration and atrophy or a suppurative destruction of the vessel, when the inflammation terminates in induration or suppuration.

In connection with the subjects discussed under this head, we must especially refer to *capillary phlebitis*, although we must confine ourselves to little more than a recapitulation of what has been already stated.

Capillary Phlebitis, if it were a true inflammation, should rather be called *Capillary Angioitis*; but inasmuch as it does not in reality consist in an inflammation of the capillaries, neither of these names is applicable to the affection. It essentially and originally consists in no textural change of the capillaries, although it very often gives rise to their atrophy or ulcerous destruction. We have already frequently referred to it, both in the general anatomy, and in different chapters of the special anatomy. It essentially consists in a coagulation of

the blood in some portion of the capillary system, dependent on a spontaneous disease of the blood, or on its infection from some cause, and is analogous to the phlebitis induced by coagulation. This coagulum, which closes these vessels, at first appears as a dark red infarctus in the affected parenchyma, and subsequently becomes decolorised, and undergoes various metamorphoses. If it be not gradually dissolved and absorbed in a finely comminuted state into the blood, it *either* shrivels, and assumes a rusty-brown, yeast-yellow, or perfectly bleached appearance, and the whole terminates in persistent obliteration of the affected capillaries (the adhesive process), associated with atrophy of the parenchyma, which is converted into a white fibroid (cellulo-fibrous) callus; *or* it undergoes, in general with considerable rapidity, a purulent and usually acute ichorous, gangreno-ichorous septic fusion, associated with a yellow, or dirty-green, or brown discoloration. The walls of the vessels and the parenchyma participate in the same process, which finally results in the formation of a purulent or ichorous abscess.

Sometimes the coagulum, after it has become yellow, undergoes partial or entire cretification.

An exudation into the parenchyma doubtless occurs simultaneously with this process. As compared with the same process in a larger vessel, we regard it as an unessential occurrence, merely depending on the permeability of the walls of the vessels.

To these processes, considered with reference to the general condition, or to a focus of infection giving rise to it, we commonly apply the term *metastases*; or in the lungs, we term them *lobular processes*, in consequence of their usually inconsiderable extent and well marked limitation.

Around their margin we commonly find a true secondary (reactive) inflammation of the tissue, which the experienced observer may tolerably readily distinguish from the original centre of inflammation, especially in the parenchymatous structures, which—as, for instance, in the case of the lungs—undergo a peculiarly striking degeneration (hepatisation). Immediately around the inflammatory centre we not uncommonly meet with capillary hæmorrhages, and suffusions of the tissue.

They occur in all tissues, but are especially frequent in certain very vascular organs which take part in hæmatisation, as the lungs, the spleen, the kidneys, and the liver. They are principally distinguished by their generally considerable number, by the roundish or (in the case of the spleen and kidneys) the wedge-like and angular form of their central part, and from their being seated on the periphery of the above named organs.

When the affection terminates in obliteration or atrophy, pit-like, contracted depressions are formed on the organ, which are generally the more numerous the nearer the morbid changes are to the surface.

Cruveilhier also treats of a *hæmorrhagic capillary phlebitis*. In reference to this name, we will only remark that the capillaries which are the seat of what is termed capillary phlebitis, cannot give rise to hæmorrhage. In the lower extremities of aged persons suffering from dyscrasia, we meet with coagula, especially in the subcutaneous veins, which extend from the trunks into the branches, and may possibly at some spots affect the capillaries. The cellular tissue is then the seat of a spontaneously distributed suffusion; it appears sometimes to contain an extravasation of loosely coagulated blood, without any apparent degeneration of texture or cohesion; and sometimes it and its contained blood represent an apparently firm, but easily torn, friable, dark red clot. We believe that this process consists in a spontaneous coagulation occurring within, and closing the trunk of the vein and its branches, and giving rise to diffuse capillary hæmorrhages, in consequence of the impediment which is presented by the occlusion to the emptying of the capillary vessels, and is proportional to the extent of the coagulation, a phenomenon which we observe, on a small scale, in almost every phlebitis—that is to say, in the form of small ramifying hæmorrhagic centres along the occluded vein, and, as we have already remarked, around the seat of capillary phlebitis.

Excessive Deposition of the Lining Membrane.—It is an unquestionable fact, that very minute arteries undergo this form of disease, since their coats lose their transparency, and become opaque and thickened either uniformly or at particular parts; and, instead of remaining soft and flexible, become rigid and brittle, and not unfrequently ossified. We feel convinced

that a similar condition exists in the true capillaries; and it is moreover probable, that the anomaly is here less in degree, since the arterial portion of the blood is expended in the process of nutrition.

This condition, which is duly considered in the Diseases of the Arteries, is very important, since, in consequence of the thickening of the walls of the vessels, it impedes their permeability and the nutrition of the tissues; since it also induces partial occlusion, or even perfect obliteration, of the vessels; and since, finally, it predisposes the vessels to laceration. Amongst its results are atrophy of the organs, spontaneous gangrene (mummification), and hæmorrhages.

It is especially observed in the brain, in association with ossification of the trunks of the vessels at the basis cerebri, and in the uterus. On making horizontal sections through the hemispheres of the brain, and passing the finger over the cut surface, we sometimes feel roughnesses, corresponding to the exuding blood-spots, and caused by the ossification of very minute arteries.

Adventitious Products.—We have already explained the relation in which the minute and true capillary vessels stand to the different adventitious products, and the changes which these vessels undergo.

V.—ABNORMAL CONDITIONS OF THE LYMPHATIC SYSTEM.

A. *The Lymphatic Vessels.*—We shall pass over the anomalies which the great lymphatic vessel, the thoracic duct, presents at its origin, in its course, and at its mouth, and proceed at once to—

§ 1. *Anomalies of Texture.*

a. *Inflammation.*—Our anatomical knowledge of inflammation of the lymphatic vessels (*lymphangioitis*) is very deficient in several points, which is in part owing to the delicacy and inaccessibility of the lymphatic vessels, and in part to our imperfect knowledge in relation to their function, and to the extent to which they can absorb heterogeneous substances. In reference to the fine ramifications of the lymphatics, they are so far positively affected in every inflammation of the tissue,

that at their numerous points of contact with the products of the inflammatory process, they absorb morbid matters by imbibition into their cavities, and, according to circumstances, sometimes lose their permeability by the swelling of their coats or the coagulation of their contents, and sometimes present torn walls. It is on the absorption of these morbid matters that the consensual inflammatory swellings of the lymphatic glands pertaining to the inflamed organ are based. In inflammations with copious exudation into the tissue, the lymphatics undoubtedly sometimes undergo a transitory occlusion, and sometimes a persistent atrophy where the inflammation terminates in induration; when there is purulent, ichorous fusion of the tissue, they suffer a destruction corresponding to the extent of the process.

It is more easy to observe the manner in which the larger lymphatic vessels are affected by inflammation, but even here there are difficulties to which we will refer after we have considered inflammation of the lymphatic vessels in a purely anatomical point of view.

Lymphangioitis presents the following persistent signs:

a. *Injection and Reddening* of the cellular coat of the vessel; we very often find *small ecchymoses* on it, and on the inner coat. In other respects the vascularisation is very frequently insignificant, and sometimes none can be detected.

b. *Infiltration of the cellular sheath* with serous, sero-fibrinous, purulent moisture, and swelling.

The simultaneous vascularisation and infiltration of the surrounding cellular tissue are often very strongly marked; abscesses often occur at various points along the vessel.

c. *The inner coat is lustreless*, dull, villous, easily wrinkled, and at some parts presents a red or bluish-red speckled appearance from the ecchymoses seen through it.

d. *The wall of the vessel is consequently thickened*, while its coats become *loosened* in texture, easily lacerable, and removable in layers.

e. The vessel is *dilated* and *varicose*.

f. *Exudation* occurs in the form of more or less opacity, of distinct coagulated flocculi, or even of larger occluding coagula, or of pure pus, in the canal of the lymphatic vessel.

Whether the occluding coagula, like those in inflamed blood-

vessels, notwithstanding the slighter coagulability of in the lymphatics, and the lesser exudative tendency of lymph, may not sometimes be coagula derived from and not solidified exudations, cannot at present be determined.

Inflammation of the lymphatic vessels may terminate in *Obliteration* or *Suppuration*, as well as in *Resolution*.

1. The lymphatic vessel may close around a foreign body, adhering to the inner coat of the vessel and metamorphosing into a fibroid string. We have observed the thoracic lymphatic vessels in a phthisical patient, who was worn to a mere skeleton, in a state of obliteration of this nature and a conversion into a solid string.

2. The lymphatic vessel may suppurate, and this occurs so much from the interior and from the purulent matter deposited in its canal and in its coats, as from a neighbouring abscess denuding and destroying the vessel. The vessel then lies as it were in the walls of the abscess, and its contents will receive an admixture of lymph, till, in consequence of inflammation around the abscess, the vessel ceases to be permeable.

When the above described changes present themselves in a lymphatic vessel, no doubt can be entertained regarding the nature of the inflammation; but we very often meet with lymphatic vessels in a condition presenting many essential similarities with inflammation, and yet, according to our views, not actually inflamed. Thus we often find the lymphatics proceeding from the lungs, or from parenchymatous organs, or from abscesses filled with pus, dilated, varicose, dull and pilous on their inner surface; while we observe that the surrounding cellular tissue presents a congested and infiltrated appearance, as is very common in the lymphatic vessels of the hypogastric and lumbar region after delivery. There are, however, absent, on the one hand, the infiltration and swelling, giving rise to the loosening of the coats of the vessel, while, on the other, the injection and inflammation of the retroperitoneal cellular substance investing the lymphatic plexus appear in puerperal cases as an integral part of the peritonitic process. We believe that in these cases the inflammation, not, or at all events is not always, produced in the lymphatic vessel itself, but is conveyed there from the inflammatory process or abscess, whether it reach the lymphatic vessels by a direct communication, or the purulent fluid, or has exuded into their cavity; or

whether it has been taken up by lymphatics opening into the abscess;—that the dilatation of the lymphatic vessels arises from the accumulation of pus within them, since its further transmission is impeded by the swelling of the lymphatic glands;—and, finally, that the loss of lustre presented by the inner coat is induced by the loosening and fusing action of the pus.

On the other hand, it is unquestionable that a lymphatic vessel containing pus not unfrequently becomes inflamed, probably in consequence of its coats imbibing pus. The period which such an inflammation occupies is frequently a long one: this is analogous to the singularly long period of incubation, which occurs in the case of poisoned wounds, from the time of the injury to the formation of a decided inflammation of the lymphatic vessels, and to the impunity with which the lymphatic vessels can convey all varieties of ulcerous products and contagious matters, while the glands are highly affected. This indicates that the lymphatic vessels possess a very subordinate sensitiveness to the irritation produced by the contact of heterogeneous matters, especially as compared with the lymphatic glands.

The appearances presented by inflammation of the larger lymphatic vessels, are in accordance with the observations which have been already made: as in phlebitis, *inflammation of the coats of a lymphatic vessel may be the primary phenomenon*, which occasions an anomaly of its contents by exudation into the canal of the vessel, or *inflammation may be excited by the presence of a heterogeneous substance within the lymphatic vessel*.

An infection of the blood by the matter produced in the lymphatic vessel, or absorbed into it from without, and the secondary (metastatic) phenomena consequent on such an infection, are in general rare; the rarity being in proportion to the distance of the process from the central anastomosis of the lymphatics and blood-vessels, and to the number of glands through which the heterogeneous substances contained in the lymphatic vessel have to pass.

Inflammation of the lymphatic vessels is often observed along the course of an inflamed vein; this may sometimes arise through the inflammation of the common cellular bed of

the vein and the lymphatic vessel, and may sometimes be dependent on the same cause as the phlebitis, namely, the absorption of heterogeneous matter.

h. *Adventitious Products*.—These are limited to *Tubercle* and *Cancer*. Each occurs in a special form, as an adventitious mass closing the tube of the vessel, and invariably as a secondary phenomenon. In order that they may occur, there must be an absorption of softened tubercle, or of cancerous blastema, into the lymphatic vessel. The lymphatic glands act as centres of absorption of the morbid matter, occasioning tuberculosis and cancer of those organs. Tuberculous pus and the cancerous blastema coagulate with the other contents of the lymphatic vessel; the former into a yellow cheesy, the latter in a whitish, more or less brain-like (encephaloid) molecular mass, which finally closes the nodularly dilated lymphatic vessel. Lymphatic vessels plugged with tuberculous matter sometimes present thickened coats and a lardaceous infiltration, doubtless in consequence of having undergone inflammation.

We may often observe both these forms, particularly tuberculosis of the lymphatic vessels, which especially occurs in the lymphatics between the intestinal and mesenteric glands, between the different mesenteric glands, and between the latter and the glands of the lumbar plexus, in tuberculous ulceration of the intestines, in tuberculous disease of the mesenteric glands (cavities in the glands), &c.; cancer of the lymphatics especially occurs in cases of medullary cancer.

§ 2. *Anomalies of Calibre.*

A moderate dilatation of one or more lymphatics, which may either be *uniform* or *nodular (varicose)*, is by no means rare; it may be dependent on pressure or on the impermeability of some of the coils in lymphatic glands. The coats of the vessel are in these cases sometimes relaxed and attenuated, and sometimes thickened. That certain cysts and hydatids, (such, for instance, as the structures occurring in the choroid plexus of the lateral ventricles, and formerly regarded as hydatids,) consist of varicose lymphatic vessels, as is taught even in the present day, is, in our opinion, by no means proved; but this does not exclude the possibility that lymphatics may sometimes assume a bladder-like dilatation at certain spots, as, for instance,

between two pairs of valves, where they may present a constricted appearance. An extraordinary and very rare example of general dilatation of the lymphatics has been recorded by Breschet, (*Le Syst. Lymph.*, Paris, 1836,) for which he was indebted to Amussat.

Contraction of the lymphatics occurs independently in general or partial atrophy, and arises from the compression exerted by every variety of tumour. It is also manifested in the form of occlusion—obliteration.

B. *The Lymphatic Glands.*

§ 1. *Anomalies of Volume—Hypertrophy—Atrophy.*—The lymphatic glands are *abnormally enlarged* in consequence of various conditions. We have here to consider more fully the anomalies induced in these structures by hypertrophy. This condition consists in an excessive accumulation of parenchyma between the lymphatics interspersed through the gland. In the present deficient state of our knowledge in reference to the structure of lymphatic glands, and to the signification of their parenchyma, we must include under the above head all enlargements of the lymphatic glands which do not depend upon hyperæmia, inflammation, or any obvious secondary formation; although it cannot be doubted that by such a classification we are compelled to include with hypertrophies many specific alterations of the glandular parenchyma. We are as yet unacquainted with the mode of its origin, as well as with the manner in which the so-called lymphatic diathesis or habitus is induced, and with the exception of the little that is known in reference to hypertrophies generally, we are ignorant of the connection that may exist between the hypertrophied development of the lymphatic glands and the co-existing disturbances of the general organic system. We are inclined, from the little that is known in reference to the subject, to regard hypertrophy of the lymphatic glands as a secondary, symptomatic phenomenon, and not as a primary and substantive anomaly.

Hypertrophy of the lymphatic glands is most common in childhood, and until the full development of puberty; although it is not unfrequently exhibited after that period, and even in mature life. The lymphatic glands of the abdomen, of the mesentery, and of the lumbar plexus, are the most frequent

seats of hypertrophy. This condition of the lymphatic glands of young persons is very commonly associated with a hypertrophied development of other blood-forming glands, as, for instance, the thyroid gland, and more especially a highly developed spleen, obstructed involution of the thymus, a hypertrophied development of the follicular apparatus of the intestines and hypertrophy of the nervous centres. Such hypertrophies either affect the whole system generally, or one portion especially, as, for instance, the glands of the abdomen.

Abnormal smallness is the result of *atrophy*. The lymphatic glands disappear in advanced age with the symptoms of general tabes, until their presence can scarcely be detected. This atrophy either affects the gland uniformly at all points, or preponderates at certain spots where the parenchyma is entirely destroyed, leaving nothing but a white, soft, cellular, shrivelled tissue. The parenchyma which remains in diminished quantity, either at the periphery or in the centre, and presents either the appearance of a capsule or a central accumulation, very commonly acquires a dull, rusty-brown colour. It is not improbable that the gland may first be reduced to the condition of a simple lymphatic nodule, as in its primary foetal state, and that the lymphatic vessels may also subsequently become atrophied together with the capillaries.

Moreover lymphatic glands may become secondarily atrophied in consequence of inflammation, and more especially of specific inflammatory processes. A marked degree of atrophy of this kind not unfrequently affects the mesenteric glands in consequence of typhous infiltration. The parenchyma of the gland is in this case absorbed, together with the product of the process. The involution of the typhoid mesenteric glands, which ultimately degenerates into tabes, has been fully considered under intestinal typhus. A similar atrophy occurs in adventitious products, partly in consequence of mechanical pressure and partly from inflammation in the neighbourhood of the adventitious structure.

§ 2. *Anomalies of Texture.*

a. *Inflammation.*—*Inflammation of the Lymphatic Glands (Lymphadenitis)* especially when it depends on the absorption from within of heterogeneous substances into the lymphatic

vessels, is of frequent occurrence in comparison with lymphangioitis. These substances may either be inflammatory products, or different contagious or deleterious matters, originating in inflammation; a distinction on which is based the difference existing between consensual inflammation of the lumbar glands and syphilitic bubo in chancre. Substantive inflammation of a group of lymphatic glands, or of a single gland, is rarely independent of the above named modes of origin, although some exceptions present themselves in the case of various specific inflammations, more especially when of a typhous character.

The lymphatic glands far exceed the lymphatics in the readiness with which they absorb heterogeneous substances, in consequence, perhaps, of the vascularity of the former, and of the manner in which their whole structure is permeated by the blood-vessels and the lymphatics; and, lastly, in consequence of the transference of matter occurring between these two systems of vessels.

A lymphatic gland presents the following alterations when in a condition of *recent acute inflammation*.

The gland is injected, and presents various shades of redness; it is swollen, relaxed, soft, and lacerable; its tissue is uniformly permeated by a serous, fibrinous, purulent exudation, or is suffused at individual points with large quantities of the same fluid. The original redness is thus variously altered, and in some cases the gland even appears as if its reddened structure were interspersed with different spots and stripes, in consequence of the absorption at individual centres of a coagulable exudation. Considerable hæmorrhage is sometimes observed to occur in the glands during the continuance of the inflammatory process, and, in that case, they are found to present variously sized centres filled with blood in different stages of coagulation and discoloration. The cellular bed of the gland participates in the process when it exhibits any considerable degree of intensity; and the former is then injected, reddened, and infiltrated, that is to say, inflamed, and the gland becomes adherent to it. It is, moreover, not unfrequently ecchymosed by small extravasations of blood.

This inflammation *terminates* in various modes, very commonly in resolution; even large quantities of solidified exudation may be readily absorbed. It not unfrequently terminates

in *induration*, in which case the copiously deposited exudation becomes converted into a fibroid callus, whilst the glandular substance is either partially or wholly atrophied. Finally, intense inflammations may result in *suppuration of the gland*—*abscess*—*phthisis of the lymphatic gland*.

Chronic Lymphadenitis is in reality a protracted inflammation of moderate intensity, with occasional acute relapses, in which the swelling of the lymphatic gland is commonly not so considerable as might be expected, in consequence of the resorption of the exudation which occurs during the remissions. It terminates in resolution through the suspension of the processes by which it had been maintained, and not unfrequently in induration with atrophy of the glandular parenchyma. The cellular bed in which the gland lies is more or less affected by the induration.

Inflammation of a gland, or of a group of glands, may readily give rise to inflammation of the adjacent glands, by the transmission of the products taken up by resorption; in this case, however, even if there is suppuration of the glands, it is seldom that the blood becomes so affected as to lead to a fatal termination.

As inflammations with a special product we must especially notice *inflammation with tuberculous exudation* and *typhous inflammation*. We shall treat of the former in our remarks on adventitious products; and the latter is fully described in our observations on Intestinal Typhus. We must, however, here especially notice the following points:

Typhous inflammation of the lymphatic glands occurs amongst us [at Vienna] as inflammation of the mesenteric glands, associated with the typhous process on the mucous membrane of the ileum, or, strictly speaking, on its follicular apparatus. It forms an integral part of ileotyphus. It particularly attacks the chain of lymphatic glands which corresponds to the affected part of the intestine, and extends from the lowest portion of the ileum till it implicates the glands of the lumbar plexus. We have here the very important question to decide—Is the inflammation of the mesenteric glands, which occurs in ileotyphus, and is characterised by its peculiar product, an affection dependant on and secondary to that of the intestinal follicles, like the inflammation of the lymphatic

glands in chancre and in certain diseases of the scalp, or is it an independent and substantive localisation of the general process?

The former view would seem to derive confirmation from this circumstance alone, that the typhous disease of the mesenteric glands is, at first, subordinate in intensity to the degree of disease affecting the follicular apparatus, and that the typhous metamorphosis of the mesenteric glands only in exceptional cases precedes that of the typhous structure in the intestine. The latter view of the question is, on the other hand, supported by several circumstances.

a. The typhous matter cannot be traced in the lymphatics during its transference from the intestine to the lymphatic glands, nor from its coagulability does it admit of such a transference in the more intense degrees of typhus; but, notwithstanding this, the mesenteric glands are already infiltrated before the loosening and softening of the typhous plaque in the intestine.

b. In anomalous forms of typhus, the mesenteric glands are obviously in a typhous condition (together with the spleen), although indeed in an inconsiderable degree, while the contiguous intestinal mucous membrane is entirely exempt.

c. In many of the more rare cases, the local process extends with excessive intensity to the mesenteric glands, without in any way affecting the mucous membrane of the intestine.

d. In bronchial typhus, the mucous membrane of the bronchi, like that of the intestines, is entirely exempt from the production of the typhous structure; for, while the typhous process is here limited to the stage of congestion and typhous catarrh, it is exhibited, in a very highly developed form, in the bronchial glands.

e. In the Oriental Plague, even the lymphatic glands, which are in no way connected with the mucous membranes, (as, for instance, with that of the intestines,) are diseased.

We are led to conclude, from the above considerations, that the disease of the mesenteric glands in ileo-typhus is a substantive affection, on the one hand from the near relation exhibited by our own typhus to the lymphatic system—a relation which is so obviously manifested in the highly developed form of plague; and on the other, from the affinity

between the true follicular apparatus of the intestines and the lymphatic system.

Next in order to typhous inflammation of the lymphatic glands, we proceed to consider—

b. Acute Swellings of the Lymphatic Glands.—These are morbid conditions of the lymphatic glands (more especially those of the mesentery), which occur in the form of acute intumescence, associated with some degree of vascularity, and with loosening of the tissue. Although these conditions very probably differ considerably in their inner character, we are as yet but very imperfectly acquainted with their nature; and hence we are able to do little more than refer to them under the above designations, which are borrowed from the most striking appearances which they present. However nearly they may seem, at first sight, to be allied to hypertrophies, for which they are very generally mistaken, they are yet very different. Inasmuch as they are developed in an acute form, and always occur simultaneously with acute diseases, which are essentially manifested as dyscrasiæ and neuroses, we think they must be regarded as the localisation of a general process of disease, and that the structure on which the increased volume of the gland depends is of a specific character. This circumstance forms the basis of the indication correctly deduced from these appearances, that they cannot be regarded as pre-existing developments connected with a chronic anomaly of the general condition of the organism, but must rather be considered to refer to an acute dyscrasia. On this account we have noticed them next in order to typhous inflammations of the lymphatic glands. They commonly, or at all events most strikingly, affect the mesenteric glands; and here, as in typhus, the follicular apparatus of the intestine is almost invariably diseased in a similar manner.

If we pass over the swelling of the glands observed in Asiatic cholera, and which is explained, provisionally at all events, by the tumultuous hyperæmia and formation of products in the whole intestinal apparatus, we may reckon the above described forms of glandular intumescence as characteristic of acute exanthemata, such as scarlet fever and variola, and of acute convulsions, such as epilepsy, tetanus, and trismus, both in children and adults. They are further observed in nu-

merous dyscrasic, febrile, and more or less genuine typhoid conditions, and are manifested during life by a complication of symptoms, and after death by a combination of anatomical alterations.

We do not think that we are in error in reference to the above observations, although, as is obvious, everything relating to this subject is still merely conjectural.

c. Adventitious Products.—The most frequent and important of these are tubercle and cancer.

1. *Formation of Cysts.*—This is of very rare occurrence, more especially when we except the formation of cysts in the lymphatic glands in association with cancer. Varicosities of the lymphatics in the glands must not be confounded with cysts, as was formerly done, nor must they be mistaken for the apertures which are occasionally observed in the stroma of the impoverished parenchyma of atrophied glands. There is an old preparation in our Museum, in which the glands of the lumbar plexus have degenerated into tumours of the size of a pigeon's or hen's egg, and which appear like a convolution of somewhat large sacs, intermixed together, and having comparatively thick walls. It is impossible to form a correct idea of the nature of these cyst-formations.

Sacs having purulent, cheesy, and greasy contents, or which are filled with a chalky paste, and are occasionally encrusted with mortar-like walls, are obsolete abscesses—tuberculous caverns.

2. *Black Pigment.*—Large accumulations of this substance frequently occur, as is well known, in the bronchial glands. It is also occasionally found, in smaller quantities, in the mesenteric glands, and even in other lymphatic glands. The bronchial glands are often so swollen with this substance, that they appear like considerable, inky, tough tumours. It is the residuum of the hæmatin, which has been deposited in the course of hyperæmia and inflammation of the glands. The blackness of the bronchial glands is associated with the well known accumulation of pigment in the parenchyma of the lungs, and it is supposed that a part of the pigment here formed is absorbed by the lymphatics, and deposited within the bronchial glands. The black colour of the mesenteric glands coincides with the discoloration of the mucous membrane round the apices,

and suppurated portions of the adjacent parts of the surface and surrounding cellular-tissue, and with the increased concentration of the lymphatic fluid in a sufficiently marked degree to produce a swelling and inflammation.

3. *Encephalitis*.—*Encephalitis* is the lymphatic glands a text-book name for all cases and the lymphatic glands the most frequent source of encephalitis disease, and more especially affects some portions of the central nervous system, as the brain and the medulla spinalis, and some of the roots and spinal ganglia. *Encephalitis* is sometimes limited to one infection.

Encephalitis leads to the following state of reference to the form in which disease occurs in the lymphatic glands.

1. *Sometimes* we find clusters, through the lymphatic glands, consisting of granules, together, or abundant, rounded, granular, semi-transparent or opaque, masses of the size of a pin's head or smaller. Some of these masses present a yellow colour and a granular texture. There is no doubt that this is the same structure as which, in other places, and especially in the lungs, we apply the name of *grey* or *white* tuberculous granulations.

2. *In other cases*, and more frequently, the lymphatic glands are changed to very large rounded or irregular, whitish, grey, or greyish, granular-masses, hard, although brittle, and giving, under pressure, rising masses, into which the lymphatic glands appear to have actually degenerated. The glands in this case form tumours, which vary from the size of a hazelnut to that of a hen's egg, or may be even larger. The granular substance sometimes, and most commonly, surrounds the adventitious matter, forming, as it were, a capsule round it; while sometimes it traverses the latter in the form of stripes, in considerable accumulations. In each form it sometimes appears vascularised, relaxed, and here and there penetrated by one or other of the above named granulations or smaller yellow, caseous masses, and sometimes it is thickened, indurated, or atrophied. We observe swollen lymphatic glands, arranged like a knotted rope along the jugular veins in the neck, in the mesentery, and along the trunks of the vessels on the lumbar vertebrae, crowded over one another into nodular heaps around the cysterna lumbalis, the head of the pancreas, the biliary ducts, the bronchi, &c.

The question now arises, whether these two forms are only various stages or different degrees of development of one and the same adventitious product. We are convinced that, in the lymphatic glands, in the same manner as in other parenchymatous structures—as, for instance, the lungs—the grey tuberculous granulations may be so accumulated, that they at length run together into considerable and apparently homogeneous masses, and, as they become yellow, constitute the cheesy nodules which are described under 2. But we do not believe that this view holds good for all cases; we are rather of opinion that the tubercle of the second form is the (tuberculous) product of an inflammation of the lymphatic glands. In favour of this view we may notice:

1. The analogy of the adventitious mass with the tuberculous exudation in other tissues.

2. The coincidence of this tuberculosis with tuberculous inflammation—as, for instance, tuberculosis of the mesenteric glands with tuberculous inflammation of the follicular apparatus of the intestines; tuberculosis of the bronchial glands, with a similar disease of the bronchial mucous membrane, with pneumonic pulmonary tubercle, &c.

3. The homogeneous character of the adventitious product throughout its mass, as well as—

4. In a large number of lymphatic glands, in addition to the absence of tubercle, in the form of grey granulations, or at most to its presence in very inconsiderable quantity.

5. Finally, the painfulness of the diseased gland, at all events at the beginning, and the accompanying fever.

Each of these forms of tubercle of the lymphatic glands not unfrequently undergoes the softening metamorphosis, giving rise to *tuberculous caverns and ulcers of the lymphatic glands, or tuberculous phthisis of the lymphatic glands*. The caverns, according to their position, open into the serous cavities, into the intestinal canal, or into the bronchi, in cases of mesenteric or bronchial glandular disease, and very often externally after suppuration of the general investments, as in tuberculosis of the cervical glands.

Sometimes, and especially in the bronchial and mesenteric glands, the tubercle undergoes the *process of cretification*. Its place in the gland is then occupied by a roundish, solid, or

partially hollow, always uneven, nodular, tuberoso, often ramifying concretion, corresponding to it in size, form, and arrangement; this concretion is often enclosed as in a capsule, and traversed by atrophied, callous, indurated parenchyma, or by a portion of gland still capable of performing its normal functions.

Tuberculosis of the lymphatic glands may be *primary*, in which case it is either confined to a certain section of the system—as, for instance, the mesenteric glands, or is diffused over nearly the whole system—as, for instance, the glands of the body generally. In the first case it is not unfrequently perfectly *isolated* and *independent*, although it is more commonly associated with tuberculosis of the organs in the immediate vicinity of the diseased glands; thus, for instance, we have tuberculosis of the mesenteric glands with tuberculosis of the small intestine, and tuberculosis of the bronchial glands with tuberculosis of the bronchial mucous membrane and the lungs. We regard this combination in most cases, and especially in intense cases, as an *original one*,—that is to say, we believe that the two structures, as, for instance, the intestinal mucous membrane and the mesenteric glands, are simultaneously affected.

Or the tuberculosis of the lymphatic glands may be *secondary*, and *dependent* on tuberculosis of some other organ. The above named combinations may serve as illustrations; thus, for instance, tuberculosis of the mesenteric glands is often associated with tubercle, and especially with ulcerating tubercle of the intestinal mucous membrane. In such cases we see the lymphatics proceeding from the intestine, and especially from the seat of the ulcer, filled with yellow cheesy tuberculous matter.

Tuberculosis of the lymphatic glands frequently acts as a starting point for other tubercloses, and especially for those of the serous membranes.

With the exception of pulmonary tuberculosis, when limited to the apices of the lungs, no tuberculosis becomes healed so frequently as the form we are now considering, when limited to one of the smaller groups of glands. The cure is effected by the suppuration (phthisis) of the gland, and the discharge of the pus externally, (as, for instance, in the neck,) or by

cretafaction of the tubercle, (as, for instance, in the bronchial and mesenteric glands.)

When, however, tuberculosis of the lymphatic glands is very widely diffused, it may prove fatal, either of itself, or in connection with other pre-existing, simultaneous, or consecutive tuberculosis through tabes, before undergoing the above named metamorphoses.

The special *seat* of tuberculosis of the lymphatic glands—regarding it as a secretion from the capillary vessels—is the parenchymatous structure; it is, however, not improbable that the second form of tubercle is exuded into the interior of the lymphatics, and effects their occlusion. The same may happen when tuberculous masses are absorbed by the lymphatics, and transferred to the gland.

4. *Cancer* frequently occurs in the lymphatic glands, sometimes as a primary, but more commonly as a secondary formation. The *medullary* is the ordinary variety, either in its genuine white form, or in association with melanosis; or sometimes combined with areolar cancer, or with cysts.

In the *primary* form, it especially attacks the glands of the lumbar plexus, and those in the mediastina; in both these positions it forms considerable tuberous growths, which in the former are known as retroperitoneal masses (Lobstein). Next in order of frequency, it occurs in the axillary, lumbar, and cervical glands. It is extremely probable that many of the cancerous structures imbedded in the cellular substance, and in which no starting point from any other definite organ can be detected, on account of the integrity of the surrounding parts, originated in one or more lymphatic glands.

It appears in a secondary form when it does not develop itself in the lymphatic glands of a parenchymatous organ until that structure has already been affected with cancer. This cancer is sometimes *very rapidly* developed. In these cases the cancerous product is always distributed over a large, generally over the greatest, part of the system; it is also usually combined with acute cancer in other organs, especially the lungs and spleen.

The *seat* of the cancer is the parenchyma of the gland; but, at all events, in cases of secondary cancer, where the disease has been occasioned by the absorption of cancerous matter

into the lymphatics, the cancer may also be seated in the lymphatic vessels of the gland.

5. *Entozoa*.—We must here notice the animal found by Treutler in the bronchial glands, and named by Rudolphi, *Filaria hominis bronchialis*.

§ 3. *Anomalies of Contents*.

These have been already noticed in the preceding pages. We may add that, once in a medico-legal examination of the body of a man, aged about 35 years, the cause of whose death was unknown, but who probably died in convulsions, and in whose intestinal canal there had been a considerable development of gas, we found several of the mesenteric glands and of the lymphatics proceeding from the intestine in a state of emphysematous inflation, which we were the more inclined to attribute to the absorption of the intestinal gas, seeing that the character and appearance of the body generally were opposed to the view that there had been a development of gas as a consequence of putrefaction.

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